

# OCCUPATIONAL DISEASES

## A Guide To Their Recognition

U.S. DEPARTMENT OF HEALTH, EDUCATION, AND WELFARE  
Public Health Service

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# OCCUPATIONAL DISEASES

A Guide  
To Their Recognition

Rewritten and Enlarged Edition of  
*Occupation Hazards and Diagnostic Signs*

W. M. GAFAFER, D. Sc., Editor  
Division of Occupational Health

U.S. DEPARTMENT OF HEALTH,  
EDUCATION, AND WELFARE

Public Health Service

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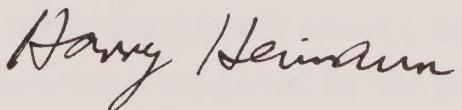
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## • foreword

The tremendous technologic achievements of the past few decades have greatly intensified the problem of occupational disease detection and control. Indeed, few, if any, of the vast array of new products and processes created by a dynamic technology are without their potential health hazards. As part of the effort to produce new tools to facilitate the discovery and prevention of job-related illness, this guide is offered to those responsible for meeting the growing challenges of occupational health.



HARRY HEIMANN, M.D.

*Chief, Division of Occupational Health*



## • preface

In 1918 the Metropolitan Life Insurance Company issued a small pamphlet entitled *Occupation Hazards and Diagnostic Signs*. The pamphlet was prepared as a guide for medical examiners and pointed out impairments to be looked for in hazardous occupations. The company revised the pamphlet in 1921, and subsequently the U.S. Department of Labor assumed the responsibility of publishing further revisions prepared by the company. The pamphlet was last revised in 1942 and the final reprinting published in 1951.

Because of the popularity of the pamphlet, the U.S. Department of Labor recognized the need for further revision when the supply of the final reprinting became exhausted. This need, as well as the medical nature of the work, prompted the Department to request the Division of Occupational Health, Public Health Service, to undertake the preparation of a revision. This has been done.

A comparison of the present work with its progenitor will reveal a number of changes in material and method of presentation suggested by the passage of almost a half century. These changes, in turn, suggested the selection of a new title which it is believed reflects more adequately the nature of the contents of the book. It is hoped that the current edition will be found even more helpful to physicians and members of allied professions engaged in efforts to prevent and control the potential diseases of the occupational environment, thus leading to the fulfillment of the primary objective of optimal health for the working population.

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# • section I

## INTRODUCTION

That occupation is recognized as an important factor in determining the cause of disability and even death is reflected in the continued revision of workmen's compensation laws to include increasing numbers of occupational diseases.

Numerous occupations involve exposures to chemical, physical, and biologic agents and there is little question that the dynamic growth of the environmental spectrum is increasing the complexity of this situation. This dynamic growth involves essentially the introduction into the environment of new processes and materials which augment or replace those previously in use. Furthermore, the size of the occupational hazards problem is increased by the fact that only a small percentage of the American working population has access to in-plant industrial and hygienic services.

### *Occupational History in Diagnosis*

Physicians are regularly consulted by patients with signs and symptoms of definite as well as indefinite character. In some instances, presenting complaints may stem from the occupation. When a physician's knowledge and interest lead him to suspect the occupation as a possible causative factor, many obscure cases can be diagnosed which previously had puzzled competent clinicians.

In perplexing cases which appear to defy diagnosis, the clinician must exert great care in determining whether any of the usual diagnostic signs known to be inherent in an occupational disease are in evidence in his patient. Of no less importance are the physical signs and symptoms of vague character which could easily be passed over unless the occupational background of the patient has been explored.

There are also situations where exposures to certain chemicals and other environmental hazards are only occasionally experienced by the worker. In such instances, there is not only an increase in risk because the danger is not suspected but also an increase in the difficulty of arriving at an acceptable diagnosis of the illness.

## 2 • OCCUPATIONAL DISEASES

The examiner must, therefore, be wholly mindful not only of the present occupation, but of former ones as well, since a patient suffering from certain ailments may no longer be engaged in the occupation responsible for his present condition.

By continued vigilance regarding the occupational history and the hazards encountered, the medical profession can use the occupational findings more effectively in forming judgments concerning disabilities as well as in the diagnosis and treatment of disease. In this way, the physician may not only add to the knowledge of occupational diseases and disabilities but also to the understanding of the possible part played by work factors in the development or aggravation of the diseases and disabilities not usually associated with the work environment. Thus, heart disease is becoming prominent among the claims submitted to workmen's compensation agencies. In such instances, the difficult problem of causal relation and disability evaluation confronts the physician.

### *Nonoccupational History*

It must be pointed out that in evaluating signs and symptoms it is essential that the physician consider also the possible part played by the non-occupational environment. Thus, the patient may have taken a medicament, in some instances momentarily forgotten, which might account for the illness. On the other hand, the patient may have chosen a hobby which he engages in after work hours in the home or the garden and involves the handling of an injurious agent which might be the offending one.

Moreover, the physician in his study of the nonoccupational environment of his patient may find a factor possibly synergistic, or potentiating, in its effect on the hazards presented by the occupation of his patient. Questions concerning the nonoccupational environment should be routinely raised; in some cases, the information elicited will be helpful in establishing a diagnosis.

### *Bases for a Diagnosis*

In any case, regardless of whether the environment concerned is occupational or nonoccupational the diagnosis must be based on (1) a meticulously taken history, (2) knowledge of the nature and severity of the exposure, (3) signs and symptoms furnishing corroborative evidence as to its accuracy, and (4) supporting clinical and analytical laboratory tests indicating the extent of the exposure.

### *Purpose and Origin*

This handbook has been prepared to assist physicians in general practice, consultants, industrial hygienists, and allied professional personnel who come into close contact with those engaged in industrial and agricultural pursuits.

Industry may find the material useful in developing its preventive health work; labor may discover much of value in its efforts to promote healthful

work environments; and those responsible for the administration of workmen's compensation should gain information helpful in the settling of claims.

The book is an outgrowth of the pamphlet, *Occupation Hazards and Diagnostic Signs*—see preface—which for many years has enjoyed great popularity and proved to be a useful reference source, as well as a teaching aid, in clinical medicine in general and occupational diseases in particular.

## Contents

The current work entailed the development of three major categories of hazard: chemical, physical, and biologic. Numerous publications, and the files of the Division of Occupational Health, served as reference sources.

The special diagnostic tests suggested under the various chemical hazards are intended as an aid to the reader with the hope that they will stimulate the use of more detailed textbook material dealing with the test or disease in question. Recommended threshold limit values are included if they have been published. It must be kept in mind, however, that these values are only suggested guidelines subject to change as evidenced by their annual publication.

Occupations associated with different environmental agents appear in various sections under the heading *Potential Occupational Exposures*. The word potential is used because it is not to be assumed that the mere presence of an injurious agent will lead to an occupational disease or disability. Much depends upon such factors as severity and duration of exposure, individual susceptibility, and the health protection practices adopted by management. Occupation information was derived principally from material made available by the Bureau of Labor Standards, U. S. Department of Labor; the records of the Division of Occupational Health; and the following books: *The Condensed Chemical Dictionary*, *Encyclopedia of Chemical Technology* and *The Merck Index of Chemicals and Drugs*. When similar activities were performed in the same or different industries efforts were made to use the same name for the occupation. In general, the term worker includes both maker and user.

Basic material appears in the sections on *Means of Contact of Toxic Agents* and *Mode of Action of Toxic Substances*. Because of their importance in occupational health, separate sections have been prepared on skin irritants and sensitizers, pneumoconioses, and pesticides. Plastics and synthetic resins are also given separately not only because this method facilitates presentation but because of the relatively numerous chemicals and hazards involved in their production. Plant and wood hazards appear separately.

A section is included which lists sources of consultation on matters pertaining to industrial hygiene and occupational health.

A list of general references useful to those interested in occupational health comprises a section. Specific references are subjoined to various sections, subsections and the different chemical hazards. It is hoped that the reader

will find this source material helpful in the further study of particular problems that may come to his attention.

### *Exclusions*

Material on treatment has not been included since such information is readily available elsewhere. The prevention and control of health hazards has been given only minor attention because it was felt that this field was adequately covered in an earlier publication, *Occupational Health Hazards, their Evaluation and Control*. The important area of workmen's compensation laws is not dealt with since material on the subject appears in periodically revised bulletins prepared by the U. S. Department of Labor under the title, *State Workmen's Compensation Laws*.

Much thought was given to the inclusion of a section on mental illness in industry since it is well known that mental disorders can complicate occupational disease, accidents, prolonged disability, work efficiency, and job stability. An example in this category is the syndrome labelled traumatic neurosis, a condition representing a post-traumatic episode which is a sequel of severe physical injury or threat of such injury, or of illness.

Mental illnesses are less easily recognized by the practitioner and allied personnel in the field of occupational health. The causes are generally obscure and the diagnosis and management of the disorder are frequently dependent upon one skilled in psychiatry. Thus, it was not possible to present the subject material in a manner paralleling the other more readily defined entities covered, except in the specific instances where acute or chronic mental disease is part of a pathologic response to an environmental factor. Omitting such a section, therefore, is not because of a lack of importance. Rather, it is felt that the subject merits far more detail than can be given in a work of this type.

### *Misuse of Information*

Since this publication has been prepared primarily as a reference source for professional personnel interested in the prevention, diagnosis, and management of occupational diseases, it is probable that some readers will encounter areas of little interest and will prefer to exercise their prerogative of judicious skipping. Yet, it is felt necessary to conclude with a word of caution lest there be made a gross misuse of the clinical data as presented. Nonprofessional interpretation of the clinical material must never become a substitute for competent medical consultation.

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## • section II

### MEANS OF CONTACT AND ENTRY OF TOXIC AGENTS

HERBERT E. STOKINGER, PH. D.

Of the various means of body exposure, skin contact is of first importance in the number of affections occupationally related. Intake by inhalation ranks second, while oral intake is generally of minor importance except as it becomes a part of the intake by inhalation or when an exceptionally toxic agent is involved. For some materials, as might be inferred, there are multiple routes of entry.

#### Skin Contact

Upon contact of an industrial agent with the skin, four actions are possible: (1) The skin and its associated film of lipid and sweat may act as an effective barrier which the agent cannot disturb, injure or penetrate; (2) the agent may react with the skin surfaces and cause primary irritation; (3) the agent may penetrate the skin, conjugate with tissue protein and effect skin sensitization; and (4) the agent may penetrate the skin through the folliculo-sebaceous route, enter the blood stream and act as a systemic poison.

The skin, however, is normally an effective barrier for protection of underlying body tissues, and relatively few substances are absorbed through this barrier in dangerous amounts. Yet serious and even fatal poisonings can occur from short exposures of skin areas, not necessarily large, to strong concentrations of extremely toxic substances such as parathion and related organic phosphates, tetraethyl lead, aniline and hydrocyanic acid. Moreover, the skin as a means of contact may also be important when an extremely toxic agent penetrates body surfaces from flying objects or through skin lacerations or open wounds.

#### Inhalation

The respiratory tract is by far the most important means by which injurious substances enter the body. The great majority of occupational poisonings that affect the internal structures of the body result from breathing air-borne substances. These substances lodging in the lungs or other parts of the

respiratory tract may affect this system, or pass from the lungs to other organ systems by way of the blood, lymph, or phagocytic cells. The type and severity of the action of toxic substances depend on the nature of the substance, the amounts absorbed, the rate of absorption, individual susceptibility, and many other factors.

The relatively enormous lung-surface area (90 square meters total surface, 70 square meters alveolar surface), together with the capillary network surface (140 square meters) with its continuous blood flow, presents to toxic substances an extraordinary leaching action that makes for an extremely rapid rate of absorption of many substances from the lungs. Despite this action, there are several industrially important substances that resist solubilization by the blood or phagocytic removal by combining firmly with the components of lung tissue. Such substances include silica, beryllium, thorium, and tolylene diisocyanate. In instances of resistance to solubilization or removal, irritation, inflammation, fibrosis, malignant change, and allergic sensitization may result.

Reference is made in the following material to various air-borne substances and to some of their biologic aspects.

### *Particulate Matter: Dust, Fume, Mist, and Fog*

*Dust* is composed of solid particulates generated by grinding, crushing, impact, detonation, decrepitation, or other forms of energy resulting in attrition of organic or inorganic materials such as rock, metal, coal, wood, and grain. Dusts do not tend to flocculate except under electrostatic forces; they do not diffuse in air but settle under the influence of gravity. Examples are silica dust and coal dust.

*Fume* is composed of solid particles generated by condensation from the gaseous state, as from volatilization from molten metals, and often accompanied by oxidation. A fume tends to aggregate and coalesce into chains or clumps. The particle diameter is less than 1 micron. Examples are lead vapor on cooling in the atmosphere; and uranium hexafluoride ( $UF_6$ ) which sublimes as a vapor, hydrolyzes, and oxidizes to produce a fume of uranium oxyfluoride ( $UO_2F_2$ ).

*Mist* is composed of suspended liquid droplets generated by condensation from the gaseous to the liquid state as by atomizing, foaming, or splashing. Examples are oil mists, chromic acid mist, and sprayed paint.

*Fog* is composed of liquid particles of condensates whose particle size is larger than mists, usually greater than 10 microns. An example is supersaturation of water vapor in air.

### *Gas and Vapor*

A *gas* is a formless fluid occupying completely the space of an enclosure and which can be changed to the liquid or solid state by the combined effect of increased pressure and decreased temperature. Examples are carbon mon-

oxide and hydrogen sulfide. An *aerosol* is a dispersion of a particulate in a gaseous medium while *smoke* is a gaseous product of combustion, rendered visible by the presence of particulate carbonaceous matter.

A *vapor* is the gaseous form of a substance which is normally in the liquid or solid state and which can be transformed to these states either by increasing the pressure or decreasing the temperature. Examples are carbon disulfide, gasoline, naphthalene, and iodine.

### *Biologic Aspects of Particulate Matter*

Size and surface area of particulate matter play an important role in occupational lung disease, especially the pneumoconioses. The particle diameter associated with the most injurious response is believed to be less than 1 micron; larger particles either do not remain suspended in the air sufficiently long to be inhaled or, if inhaled, cannot negotiate the tortuous passages of the upper respiratory tract. Smaller particles, moreover, tend to be more injurious than larger particles for other reasons. Upon inhalation, a larger percentage (possibly as much as 10-fold) of the exposure concentration is deposited in the lungs from small particles than from larger particles. In addition, smaller particles appear to be less readily removed from the lungs. This additional dosage and residence time act to increase the injurious effect of a particle.

The density of the particle also influences the amount of deposition and retention of particulate matter in the lungs upon inhalation. Particles of high density behave as larger particles of smaller density on passage down the respiratory tract by virtue of the fact that their greater mass and consequent inertia tend to impact them on the walls of the upper respiratory tract. Thus, a uranium oxide particle of a density of 11, and 1 micron in diameter will behave in the respiratory tract as a particle of several microns in diameter, and thus its pulmonary deposition will be less than that of a low density particle of the same measured size.

Other factors affecting the toxicity of inhaled particulates are the rate and depth of breathing and the amount of physical activity occurring during breathing. Slow, deep respirations will tend to result in larger amounts of particulates deposited in the lungs. High physical activity will act in the same direction not only because of greater number and depth of respirations but also because of increased circulation rate, which transports the toxic agent at a greater rate to critical tissues, and the presence of increased amounts of certain hormones that act adversely on substances injurious to the lung. Environmental temperature also modifies the toxic response of inhaled materials. High temperatures in general tend to worsen the effect, as do temperatures below normal, but the magnitude of the effect is less for the latter.

*Biologic Aspects of Gases and Vapors*

The absorption and retention of inhaled gases and vapors by the body are governed by certain factors different from those that apply to particulates. Here solubility of the gas in the aqueous environment of the respiratory tract governs the depth which a gas will penetrate in the respiratory tract. Thus, very little, if any, of inhaled, highly soluble, ammonia or sulfur dioxide will reach the pulmonary alveoli, depending on concentration, whereas relatively little of insoluble ozone and carbon disulfide will be absorbed in the upper respiratory tract.

Following inhalation of a gas or vapor, the amount that is absorbed into the blood stream depends not only on the nature of the substance but more particularly on the concentration in the inhaled air, and the rate of elimination by the body. For a given gas, a limiting concentration in the blood is attained that is never exceeded no matter how long it is inhaled, providing the concentration of the inhaled gas in the air remains constant. For example, 100 parts per million of carbon monoxide inhaled from the air will reach an equilibrium concentration in the blood corresponding to about 13 percent of carboxyhemoglobin in 4 to 6 hours. No additional amount of breathing carbon monoxide will increase the blood carbon monoxide level. Upon raising the amount of carbon monoxide in the air, however, a new equilibrium level will eventually be reached.

### Ingestion

Poisoning by this route in industry is far less common than by inhalation for the reason that the frequency and degree of contact with toxic agents from material on the hands, food, and cigarettes are far less than by inhalation. Because of this, only the most highly toxic substances are of concern by ingestion. Examples are lead, arsenic, and mercury.

The ingestion route, however, passively contributes to the intake of toxic substances by inhalation. That portion of the inhaled material that lodges in the upper respiratory tract is swept up the tract by ciliary action and is subsequently swallowed, thereby contributing to the body intake.

The absorption of a toxic substance from the gastrointestinal tract into the blood is commonly far from complete, despite the fact that, in passing through the stomach, substances are subjected to relatively high acidity and, on passing through the intestine, are subjected to alkaline media.

On the other hand, favoring low absorption are observations such as the following: (1) Food and liquid mixed with the toxic substance not only provide dilution but also reduce absorption because of the formation of insoluble material resulting from the combinatory action of substances

commonly contained in such food and liquid; (2) there is a certain selectivity in absorption through the intestine that tends to prevent absorption of "unnatural" substances or to limit the amount absorbed; and (3) following absorption into the blood stream, the toxic material goes directly to the liver, which metabolically alters, degrades, and detoxifies most substances.



## • section III

### MODE OF ACTION OF TOXIC SUBSTANCES

HERBERT E. STOKINGER, PH. D.

Toxic substances exert their effects by physical, or by chemical or physiologic (enzymatic) means, or by a combination of both.

The classification as presented here of the toxic mechanisms in the mammalian host has no precedent or any accepted basis other than that it appears to be inclusive, reasonable, and practicable. The classification has been developed to delineate two basic actions: the action of the toxic substance on the host, and the action of the host on the toxic substance. For it is the interplay of these two actions, together with the rate at which the body excretes the toxic substance, that determines what is called the toxicity of a substance.

Thus, the full toxic potential of most substances is not usually asserted, because of destructive actions by the body and its mechanisms of elimination by urine, sweat, feces, and exhalations, or because of sequestration in inactive forms at certain tissue sites such as bone, skin, hair, and nails. If this were not so, synergistic or enhanced toxicities would never be manifest. Synergistic or enhanced toxicities arise from the development of unusual or enhanced concentrations of the toxic substance. This occurs when one or more of the usual means of elimination or reduction of the toxic substance are blocked.

It is to be recognized that the following classification on toxic mechanisms must necessarily be based on prevailing knowledge, which varies greatly from discipline to discipline. In enzymology, for example, the state of knowledge is at the molecular, and in some instances, at the submolecular level. Such a situation obviously permits more exact definition of the governing mechanisms than is afforded by a discipline in which knowledge is at a cellular or organ level. Thus, a mechanism regarded at present as physical might be later labelled chemical or enzymatic to reflect the acquisition of new knowledge at a more intimate level. Indeed, when all mechanisms can be explained at the submolecular level, an entirely different classification will result. It is hoped that the classification, believed appropriately designated

within the limits of present knowledge, may not only provide greater insight into how chemicals act in the body but also point to possibly unsuspected relationships among the actions of diverse chemicals.

### Physical Modes of Action

Harmful substances that have a solvent or emulsifying action can produce, after prolonged or repeated contact, a dry, scaly, and fissured dermatitis. This effect is commonly attributed to the physical removal of surface lipid, but may also be caused by denaturation of the keratin or injury to the water barrier layer of the skin. Acidic or alkaline soluble gases, vapors, and liquids, may dissolve in the aqueous protective film of the eye and mucous membranes of the nose and throat, and in sweat, causing irritation at these sites. Moreover, such insults may erode teeth and produce changes in hair structure.

On the inner surfaces of the body, the lungs and gastrointestinal tract, physical contact of unphysiologic amounts of substances causes irritation. This may lead to inflammation, or produce contraction, as in the reflex constriction of the respiratory passages upon inhalation of an irritant gas with resultant coughing, choking, or asphyxiation. In the upper gastrointestinal tract, the effect may include vomiting and, further down in the tract, the irritation may result in peristalsis and defecation.

Inert gases can exert serious and often fatal effects simply by physical displacement of oxygen, leading to asphyxia. Under pressure, inert gases such as nitrogen can produce compressed air illness by dissolving in unphysiologic amounts in the blood, lymph, and intercellular spaces, or may rupture delicate membranes such as the eardrum. Sudden, or too rapid, decrease in pressure results in decompression sickness. Less inert gases such as carbon dioxide and oxygen under greater than atmospheric pressure can lead to narcosis and other more serious effects, such as nerve and brain damage.

Physical adsorption of gases or vapors on solid or liquid particulates (aerosols), may, upon inhalation, lead to physiologic effects out of proportion to that anticipated from their inhaled concentration prior to adsorption. The action is known as synergism when the effect of gas and particulate exceeds the sum of the effects expected from either alone, or antagonism when the effect is less than expected. A physical theory has been developed to explain these abnormal actions. It is based on molecular properties of gases, and accounts for the synergism, by postulating "adsorbed" layers of the gas on the particulate that, upon inhalation, carry to the sensitive lung tissue enormously increased concentrations of the gas that become localized point sources of contact. Synergism results when a rapid rate of desorption of the gas from particulate to the tissue occurs; antagonism, when the desorption rate is very slow or nonexistent.

An example of synergism is the inhalation of a mixture of sulfur dioxide and sodium chloride crystals in which the effects on broncho-constriction are greater than that from the same concentration of inhaled gas. Sodium chloride inhaled alone is inert. An example of antagonism is the inhalation of welding fumes of nitrogen oxides and iron oxides; reduction of effect in this case is explained on the basis of a firmly *combined* layer of nitrogen oxides on the iron oxide particles.

Radioactive particles cause dislocation and breaking of chromosomal linkages apparently from local energy release.

### Chemical or Physiologic Modes of Action

Substances that act chemically to produce injurious effects on the organs and tissues of the body do so by two basic means, either by depression or stimulation of normally functioning pathways of metabolism. These two effects are brought about by a variety of mechanisms that are known in only a general way for most toxic substances. But there are a few important substances such as carbon monoxide, cyanide, arsenic, and uranium, for which detailed mechanisms are known. In other cases, mechanisms are but partially known.

It is possible also that a single substance may have more than one pathway of action, or act by stimulation of an enzyme system at a low concentration of the substance and by depression at a higher concentration. This is a characteristic response of many, if not all, toxic substances, better known examples of which are arsenic, cobalt, vanadium, chloroform, and benzene.

It is convenient to consider chemical mechanisms under the following categories: (1) Primary mechanisms of injury which involve interactions of the toxic substance at the enzymatic level; (2) nonenzymatic interactions which involve more or less direct chemical combination or replacement of the toxic substances with a body constituent without enzyme intervention; and (3) secondary mechanisms of injury that may involve both enzymatic and nonenzymatic actions resulting in injury only indirectly as a consequence of the presence of the toxic substance.

#### *Primary Enzymatic Mechanisms*

Most of the metabolic activity of the body is a result of the activity of enzymes, biologic *catalysts* formed by living cells throughout the body. Consequently, it is reasonable that the bulk of all toxic mechanisms should involve interference in some way with normal enzyme activity.

Enzymatic actions occur throughout the body without restriction to any particular organ site, although the liver cells perform a major proportion of the metabolic activity of the body. Equally active, however, but less diversified, are all other tissues in the body, the lung, kidney, intestine, brain and nervous tissue, and bone as well. From this it may be inferred that

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enzymatic mechanisms may occur with the enzyme situated at nerve endings, within the nerve cell itself, or at cell surfaces.

It may be observed that two groups of enzymes, phosphatases and dehydrogenases, are commonly involved in a large variety of toxic mechanisms. The reason for this is that the two groups are included in a large number of important enzyme systems in the body.

In "metabolizing" a toxic substance, it is important to observe that the enzyme is merely performing a function that it normally performs in metabolizing natural foodstuffs; no special enzymes exist to metabolize toxic substances.

Although substances are toxic for a variety of causes, one of the causes is the frequent inability of enzymes to metabolize completely, and thus to destroy, the toxic substance. The reason for this is attributable to the rather high specificity, or selectivity, of the enzyme for the substance it is attacking, the *substrate*.

Enzymes are proteins, highly complex interlocking chains of amino acids, possessing to a marked degree specific, spatial orientation of the chemical constituents. The orientation of the enzyme is such that it fits, much like the key to the lock, the substrate with which it combines prior to modifying it.

It is now recognized that certain enzymes heretofore considered homogeneous in composition and in action may consist of several distinct components, each still acting, however, on the same substrate; these components are called *isoenzymes*.

The substrates which enzymes act on with highest efficiency are those with chemical structures and configurations of natural foodstuffs. Foreign, toxic substances do not possess these precise spatial requirements. It is thus apparent why enzymes only incompletely metabolize toxic agents.

Many enzymes have additional specificity requirements, in that they require a metal or a vitamin, or both, as *cofactor(s)* or *activator(s)*. For example, the enzyme cocarboxylase, that splits carbon dioxide from certain organic acids, requires vitamin B<sub>1</sub> and magnesium ions as necessary constituents before it can function.

Because enzymes are proteins, they exhibit the physical and chemical properties of proteins. They undergo *denaturation* (1) by heat, as in burns, (2) by marked changes in acidity or alkalinity as effected, for example, by contact with corrosive agents, or (3) by chemical denaturing agents, such as urea in high concentrations. These agents alike cause structural and configurational changes in the protein, and the characteristic specificity is lost, and with it the catalytic activity of the enzyme.

Enzymes may become inactivated to varying degrees by less drastic means, however. Among those enzymes requiring a specific metal as activator, any agent that will displace or render inactive this metal will render the enzyme

inactive to the degree that the metal was rendered inert. Certain metals with similar spatial requirements for the specific metal required by the enzyme may do this. Certain poisonous metals such as beryllium are believed to act in this way. Cyanide may combine with the iron of an iron-dependent enzyme and inactivate or *inhibit* the enzyme.

Another common way an enzyme may become inhibited is from competition with a substance whose structure is sufficiently similar to the natural substrate but does not quite fulfill the spatial requirements of the enzyme. This is probably the most common way in which toxic substances exert their effect on enzymes.

A third way by which enzyme activity is inhibited is by accumulation of the product of the enzyme's activity. This is one of the natural ways by which body enzyme activity is regulated.

Like other catalysts, enzymes theoretically undergo no net change during the reactions they catalyze. Within a minute, one molecule of an enzyme can alter many thousand molecules of the substrate (*turn-over-rate*). In no case does the enzyme contribute to the net energy requirements of the reaction and only those reactions that are energetically possible without an enzyme can occur in its presence. Enzymes merely accelerate a chemical reaction. They catalyze the backward as well as the forward direction of the reaction.

The fundamental aspects of enzyme activity with respect to toxicity may be summarized as follows. Enzymes combine with the toxic substance. This combination may be inhibited partly or completely by the toxic substance, or the enzyme may act on the toxic substance more or less incompletely, possibly with the production of even more toxic substances. If the enzyme whose activity is blocked is a critical one, the slowing down may occur of some vital function resulting in alteration of cellular constituents in amount or type, or even in cell death.

A discussion follows of presently known enzymatic mechanisms.

*Direct combination*—The simplest way by which a toxic substance can alter enzyme action is by direct combination of the substance with active groups on the enzyme structure. Such is believed to occur with certain metals as mercury and arsenic which combine so tightly with the active group of the enzyme that further action is blocked. If the enzyme or enzymes represent critical systems for which there is no shunt mechanism, then cells may die or function subnormally resulting ultimately in injury to the cell, the organ, and the host. Similarly, nonmetallic substances such as cyanide can combine with and block the action of heavy metal-bearing enzymes because of the production of an inactive metal-cyanide enzyme. The blocking of this enzyme system to a significant degree results in the well-known fatal cyanide poisoning.

Another mechanism of poisoning by direct combination is illustrated by substances such as ozone and nitrogen dioxide, and possibly iodine and

fluorine, that destroy enzymes by oxidation of their functioning groups. In these cases, specific chemical groups such as -SH and -SS- on the enzyme are believed to be converted by oxidation to nonfunctioning groups; or the oxidants may break chemical bonds in the enzyme leading to denaturation and inactivation.

One of the more commonly encountered enzyme inhibition mechanisms in occupational exposures is that of the inhibition of the action of cholinesterase (acetylcholine esterase), an enzyme that regulates nerve-muscle action by destroying the muscle excitor acetylcholine. This muscle excitor is a powerful pharmacologic substance which if not destroyed when it is free can act as a poison. The destruction is accomplished by the hydrolysis of the potential poison into its components, an acetyl group and choline. A large number of pesticides, chiefly organic phosphates, act in the body by blocking this enzyme action, thus allowing excessive amounts of the muscle stimulator to accumulate. The excessive stimulation results in paralysis and prostration.

*Competitive inhibition*—A second, and one of the more usual toxic mechanisms involving enzymes, is that of competition of the toxic substance with normal metabolites, or the cofactor(s) essential for enzyme action, for the site of action on the enzyme. This form of competition is highly effective, and thus injurious, only when the chemical structure of the competing toxic substance resembles that of the constituent normally used by the enzyme; the closer the structural similarity, the more effective the competition.

The successful competition of an unnatural or foreign toxic substance for the enzyme sites of action blocks normal action by not permitting either significant amounts of normal substances to be metabolized, or by preventing combination of a cofactor necessary for enzyme action. The cofactor can be a metal or a highly complex specific organic substance such as a vitamin.

Competitive inhibition, first shown to be the action of sulfanilamide by reason of its close similarity to the B vitamin, para-aminobenzoic acid, has been demonstrated to function similarly in many other drug actions; it is also the basis of the mechanism of action of a number of anticancer drugs, many of which are appreciably toxic, for example, the fluoropyrimidines.

Toxic mechanisms may operate also by metal-to-metal competition. For example, it is believed that the poisonous action of beryllium results from its capacity to compete effectively for the sites of combination of magnesium and manganese on critical body enzymes, by which action the enzyme is no longer able to function at its normal rate or may be inactivated completely. This competitive inhibition of foreign metals is a very general way by which metals exert their toxic action.

A highly interesting example of a competitive mechanism is that recently found to explain the increased toxicity sustained following simultaneous exposure to two structurally similar economic poisons, malathion and EPN. Although EPN is highly toxic, malathion has a far lower order of toxicity.

When the two substances are present in the body together, however, malathion has a toxicity equalling that of EPN, and the summated toxicities of both is far beyond expectation.

The explanatory mechanism is found to reside in the fact that, inasmuch as both substances have chemically similar structures, EPN effectively competes for the same enzyme that hydrolyzes and thus would otherwise reduce the toxicity of malathion. By inhibiting this enzyme action, the concentration of the toxic form of malathion is maintained at a high level in the body, and consequently the toxicity is enhanced.

This is not an isolated instance of such a competitive mechanism. A number of other combinations of economic poisons are believed to produce enhanced toxicities by similar mechanisms, for example, the combinations malathion and dipterex<sup>R</sup>, and guthion<sup>R</sup> and dipterex<sup>R</sup>. Other similar examples, but not involving the organic thiophosphates, undoubtedly will be found.

*“Lethal synthesis”*—Another means by which enzymes are involved in toxic mechanisms concerns the synthesis of a new toxic product by enzyme action on the toxic substance originally taken into the body. The newly synthesized product then exerts its toxic effect by interfering with normal metabolic processes.

A striking example of a substance involved in this type of mechanism is the rat poison 1080, sodium fluoroacetate. Following its absorption into the body, an enzyme transfers the fluorine atom in fluoroacetate to citric acid, an important intermediate in the cycle of terminal metabolism. The converted fluorocitrate, unable to function to a significant degree in this important metabolic cycle, breaks the metabolic chain of activity, with the result that tissue respiration ceases, and death ensues.

*Toxic enzymes*—A rather unusual type of toxic mechanism results when the toxic substance itself is an enzyme. A number of these instances are known. They are associated with the introduction into the body of such substances as snake and bee venoms and bacterial toxins. Although these substances exhibit a variety of toxic manifestations, the mechanisms of some of which are as yet unknown, the venoms of bees and certain snakes possess enzymes (phosphatidases) that lyse red blood cells destroying the oxygen-carrying power of the blood, as well as enzymes (proteolytic) that destroy cells and inhibit blood coagulation. In addition, bee venom contains a substance that inhibits dehydrogenases, enzymes important in the metabolism of many body functions. Snake bites are currently an occupational hazard in certain areas of the United States.

*Inducible enzymes*—Thus far all of the mechanisms discussed have been depressant in action. As indicated previously, response of toxic substances may under certain conditions act as stimulants to metabolic activity. In this category may be placed inducible (adaptive) enzymes, by which is meant the physiologic synthesis of additional amounts of an enzyme in response to the

presence of an inducing agent. In this instance, the inducing agent is a toxic substance.

Because inducible enzymes are difficult to demonstrate in the mammalian host (although a number have been so demonstrated in bacteria and yeasts), only one instance of industrial health interest is presently known in sufficient detail; undoubtedly others will be found. High sucrose diets fortified with vitamins fed for 3 weeks to rats stimulate the enzymatic production of additional amounts of protein sulfhydryl groups in the kidney, which enables the rats to withstand otherwise lethal doses of mercury. The newly-formed sulfhydryl binds the mercury firmly, thus effectively reducing its toxic potential.

A mechanism exemplifying stimulation, probably mediated through inducible enzymes, is the increased production of serum alpha globulins by cobalt when absorbed into the body at relatively low levels of intake. At slightly higher levels of intake, cobalt stimulates the production of increased amounts of red blood cells (polycythemia production); associated with the polycythemia is increased production of hemoglobin. The exact mechanism of this stimulation is not known, but a new hormone, erythropoietin, whose production is stimulated by cobalt, is believed involved. It appears also that the action of erythropoietin is not entirely restricted to stimulating bone marrow to increased production of red cells but may include stimulation of other centers as well.

### *Nonenzymatic Mechanisms*

There are a number of industrially important types of poisoning which proceed through mechanisms that do not involve the intervention of enzyme action but for which the energy is supplied, so far as is known, by chemical action.

*Direct chemical combination*—Among the best known and understood mechanisms of poisoning is that of direct chemical combination of the toxic substance and a body constituent, as illustrated by carbon monoxide poisoning. In this instance, the gas combines rapidly and rather firmly with hemoglobin forming a new compound, carboxyhemoglobin, that cannot perform the usual function of hemoglobin, which is the transport of oxygen to the tissues.

Hydrogen sulfide likewise unites with hemoglobin to convert it to sulfhemoglobin, a nonoxygen carrying pigment, although this mechanism is not important in hydrogen sulfide poisoning.

*Release of body constituents*—A less well understood mechanism of injury, but on which there is nevertheless an enormous amount of indirect evidence, is the release by toxic substances of natural body constituents in abnormal amounts that lead to injury and even death. Instances of this mechanism are numerous and involve the intake into the body of such common substances as "hay-fever" allergens or other allergenic materials, for example, tolylene diisocyanate.

Intake of these substances results in release of histamine or histamine-like substances in local large amounts with the characteristic development of inflammation, edema, and other evidences of injury. A large number of amines are capable of histamine release; in these instances the mechanism involved is believed to be one of displacement, whereby the tissue-bound histamine is displaced and liberated by the unnatural amine. Similarly, any type of simple cellular damage results in the liberation of histamine-like substances.

There is accumulating evidence also that release of hormones from nerves may be the common mechanism by which a number of chemical substances exert their toxic action. The example that follows not only illustrates an action that releases body constituents, but also illustrates a highly indirect toxic action formerly believed to be a direct effect on a substance on an end organ.

Carbon tetrachloride has been shown to cause the massive discharge of epinephrine and related neurohumors from central sympathetic nerves. This discharge possibly mediated by enzyme action results in the stimulation of the nerve supply to the blood vessels of the liver to produce (1) restriction of the liver's blood flow leading to reduced oxygen transport and, ultimately, the characteristic centri-lobular necrosis of the liver and (2) release of unesterified fatty acids from fat depots and their deposition in the liver to produce the well known "fatty" liver of carbon tetrachloride poisoning.

*Chelation*—A toxic mechanism that is increasingly being recognized to be one of the more common pathways of toxic action is chelation. Chelation is the term applied to the chemical combination of an organic structure and a metal whereby the metal is very firmly bound to the organic substance by both nonionic (organic) and ionic bonding. For example the therapeutic agent EDTA binds metals by chelation. Many drugs and antibiotics are now believed to act by chelation. By so acting, these substances exert their effects in a number of ways:

(1) By removal of biologically active metals that are normally bound in the cell or its components with resulting inactivation and cell damage. For example, treatment of lead poisoning with EDTA may in addition remove other metals such as zinc, that is required for important functions in certain kidney enzymes (carbonic anhydrase).

(2) By reacting with fixed intracellular metals.

(3) By chelating firmly with a fixed tissue constituent. This is believed to be the mechanism by which boron, as borate, exerts its toxic action. Borate is known to chelate with adjoining carbon atoms containing hydroxyl groups. If the structure prior to chelation happens to be a critical one in a metabolic chain, ordinary function ceases and injury occurs as a result of the altered chelated structure.

(4) By increasing the absorption of a toxic agent. Instances are being recognized of toxicity resulting from abnormally increased amounts of absorption into the blood stream by a chelating compound. Iron, normally nontoxic when absorbed by the usual regulatory mechanism, may under unusual circumstances be absorbed in toxic amounts by the mechanism of chelation to form a soluble, easily absorbed substance.

*Stimulation of immune mechanisms*—A mechanism whose toxic significance remains to be fully evaluated, but which nevertheless has been recognized for many years, is the stimulation of immune mechanisms as a result of the production of a new antigenic structure from the combination of a toxic substance with body constituents, usually protein. This mechanism is thought to be the basis of skin sensitivity resulting from contact with certain reactive organic substances, for example, the chloronitrobenzenes.

Another substance that illustrates this mechanism strikingly is tolylene diisocyanate and related aromatic isocyanates. These substances, upon inhalation, have unusual avidity for combining with body protein with resultant allergic sensitization of the respiratory tract.

### *Secondary Toxic Mechanisms*

In this category are grouped those pathways of metabolism and mechanisms of injury that are not effected by the direct action of the toxic substance but develop either (1) as a result of metabolic alteration of the toxic substance following its entrance into the body, or (2) as a consequence of an accumulation of toxic by-products from the initial, direct action of the toxic substance. In the second instance, further injury occurs at a site in the body different from that of the original toxic action. Most, if not all, of the mechanisms considered here are performed by enzymes.

*Detoxication (metabolic) mechanisms*—Mechanisms grouped here comprise all those metabolic activities that the *body* performs on a toxic substance in contradistinction to the actions that the *toxic substance* performs on the body. The latter actions were considered under Primary Enzymatic Mechanisms, and Nonenzymatic Mechanisms. Broadly, the so-called “detoxication” mechanisms are those performed by the body in the process of attempting to eliminate the toxic substance, namely, oxidation, reduction, and synthesis. A few examples of each of these mechanisms will be given for well-known industrial substances of a toxic nature.

It will become apparent that the body does not always act to its own advantage when handling a foreign, and generally atoxic, substance. These peculiarly disadvantageous reactions result, however, merely because the body is equipped with certain definitive pathways of metabolism derived from past utilization of food components. These are its only resources when confronted with nonfood substances, and accordingly these mechanisms are used insofar as they can act to a degree on foreign substances bearing chemical structures similar in some respects to food substances. Whether

this indiscriminate action by the body's enzymes results in an outcome favorable or unfavorable to the body depends only on the nature of the resultant modified foreign substance and not on any selective or guided action of enzymes. Some examples of *oxidation*, *reduction*, and *synthesis* follow.

(1) *Oxidation* is one of the most general metabolic activities of the body against foreign substances. It includes the oxidation of alcohols to aldehydes, aldehydes to acids, oxidation of hydrocarbon rings to phenols and quinones, alkyl groups to alcohols and acids, oxidative removal of ammonia from amines, oxidation of organic sulfur compounds, oxidative splitting of carbon ring compounds, removal of halogens from halogenated hydrocarbons, and a variety of other reactions including the oxidation of certain metallic ions.

A well-known example in which secondary oxidative mechanisms are believed to play a dominant role in the toxicity of an alcohol is that of methyl alcohol. Oxidation to formaldehyde, which subsequently interferes with oxidative enzyme synthesis, is believed to be the pathway by which methyl alcohol exerts its injurious effect on the optic nerve leading to blindness. Ethyl alcohol, and presumably other alcohols, proceed through this metabolic pathway of oxidation to the corresponding aldehyde, which is responsible, in part at least, for the toxic effects.

Perhaps one of the more important and interesting examples in which oxidative mechanisms play a decisive role in the ultimate toxic response is the oxidation of the cancerogenic hydrocarbon, 3,4-benzpyrene. Current theories of cancerogenesis consider some oxidized product, not the original hydrocarbon, to be a step in the process leading to tumor development. Several oxidized products of 3,4-benzpyrene have been identified following its entry into the body including phenolic products and several quinones.

Similarly, the serious effect of the hydrocarbon, benzene, is believed to be the result of increasing oxidation of the benzene nucleus, first to phenol (monohydroxybenzene), then to dihydroxy- and trihydroxy-phenol, which are considered responsible for the toxicity of benzene. Further oxidation to quinone may be involved, followed by further oxidative cleavage of the benzene ring to form the relatively nontoxic mucic acid.

In this connection, it should be recognized that by no means do all metabolic alterations in the structure of toxic organic substances result in toxic by-products. A sizeable number of the metabolic products are detoxified in the process, as is reasonable.

An important and striking example of the role of oxidative mechanisms in developing the toxicity of an organic substance is parathion. This substance, containing sulfur in its molecule, is relatively nontoxic until oxygen replaces the sulfur forming paraoxon which is extremely toxic, inhibiting completely an important enzyme of nerve function, cholinesterase.

An example of oxidation among inorganic toxic substances is that of uranium. The tetravalent form is unstable to the body's oxidation-reduction potential, and is oxidized to the more toxic hexavalent form. The hexavalent form then combines with active sites (phosphate groups) on the surface of cells, blocking normal metabolic processes necessary for cell survival.

Much, if not all, of the toxicity of the long-recognized poisoning action of aniline arises not from aniline itself, but from its various oxidation products formed in the body. The more important of these are para-aminophenol and, by further oxidation, the quinoneimine which is believed responsible for the methemoglobinemia that develops when aniline, or other aromatic amines, are absorbed into the body. The oxidized product of aniline oxidizes the ferrous iron of hemoglobin to the ferric form, resulting in methemoglobin, incapable of releasing oxygen.

(2) *Reduction* is far less common a body function than oxidation. Nevertheless several types of foreign organic substances are metabolized by this pathway to produce one or more substances that are more injurious than the parent substance. Among certain of the inorganic metal ions, reduction is also the pathway of metabolism. Organic nitro-groups are reduced by stages to amines. Some aldehydes are reduced to alcohols. Unsaturated double bonds of carbon compounds may add hydrogen and thus become reduced. These types are not an exhaustive listing.

In general, however, reduction, contrary to oxidation, tends to result in products that are less toxic than the original substance, for example, reduction of aldehydes to alcohols, and are thus of lesser interest here. On the other hand, metabolism of nitrobenzene results in a number of products, one of which, para-aminophenol, is from 50-80 times more acutely toxic than the parent nitrobenzene.

Among inorganic ions, pentavalent arsenic is relatively inactive in the body until reduced to the trivalent state. The physiologically active form of manganese is trivalent. If manganese is taken into the body in the form of pyrolusite in which the manganese is tetravalent, reduction to the active form must occur, at least to that portion which is absorbed into the blood stream and later incorporated into active tissue components.

(3) *Synthesis*, whereby the body contributes some tissue constituents in the conversion of the foreign substance to a new product, is one of the more common means the body has of disposing of the toxic agent. There are a dozen known synthetic mechanisms to accomplish this. Without listing them all, the addition of such substances as sulfate, sulfur, glucose, and protein derivatives to the toxic substance in general results in true detoxication and lessening of the injurious effects of the foreign substance.

The well-known synthesis of phenylsulfate, which was one of the earliest synthetic mechanisms to be discovered (1876), converts highly toxic phenol to a substance which is practically nontoxic. Cyanide, both inorganic and

organic forms, is synthesized to thiocyanate, a structure many times less toxic than cyanide. Certain toxic metal ions may react with sulfur of the body to be excreted as insoluble, and, thus nontoxic, metal sulfides.

It should be pointed out that these synthetic detoxifying mechanisms are not entirely free of injury to the body. In contributing some of its constituents, the body may deprive itself of vital amounts of these substances if synthesis is prolonged, and thus injure itself.

*Secondary organ involvement*—A secondary mechanism of very general nature, and of considerable toxicologic importance, involves the indirect action of either the toxic agent or its metabolic by-products, or both. Once having injured a primary site, the substance(s) causes either the production or accumulation of deleterious products that in turn affect a secondary site.

A striking example of this secondary mechanism is the action of hexavalent uranium, which first injures the kidney in such a way as to prevent normal elimination of waste products such as urea, ammonia, and other substances. These products accumulate in the blood stream and injure the liver, resulting in fatty degeneration of this organ.

Similar indirect injury occurs to the heart when the lung, through direct injury by some toxic substance, restricts blood flow thus placing undue stress on the heart.

There are numerous other examples; in fact, the function of the body is so organized that there are few alterations of significant magnitude in an organ or tissue site that do not have repercussions in some other organ even at a remote site. The interlocking activities of the endocrine glands, with their respective hormones and their dependence on vitamins and minerals for normal function, is the basis for this entire group of secondary mechanisms.

An interesting example of the involvement of these highly sensitive interlocking endocrine systems is the simple inhalation of nonlethal concentrations of ozone, which produces alterations in the activities of the adrenal glands and disturbs the normal uptake of iodine by the thyroid gland, which in turn alters the activity of the thyroid-stimulating hormone of the pituitary body.

### Conclusion

Reference was made to different physical modes of action of harmful substances and the effects of such substances following contact with outer and inner body surfaces.

The discussion of toxic mechanisms attempted to present as simply and briefly as possible a unified and comprehensive view of the entire field which, as is seen, is highly complex and involves most of the vital functions of the body.

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## • section IV

### OCCUPATIONAL DERMATOSES

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An occupational dermatosis is any cutaneous abnormality resulting directly from, or aggravated by, the occupational environment. Numerous indirect factors contribute to the development of an occupational dermatosis. Among them may be age, race, sex, season, the pre-existence of other skin diseases, type of skin, perspiration, and poor personal hygiene.

The term occupational dermatitis is a more restrictive one, signifying inflammation of the skin, or an eczematous process; excluded are neoplasms, infections, and certain pigmentary disturbances. Because contact is the usual means of acquiring an occupational dermatitis, the terms occupational dermatitis and contact dermatitis are used interchangeably in industry. The direct causes of contact dermatitis, chiefly chemicals and plants, may be divided into primary irritants and allergic sensitizers.

Because of their external location, the occupational dermatoses are readily accessible for diagnosis and treatment. The most important of these diseases is contact eczematous dermatitis which usually can be treated, and further attacks averted, simply by prevention of contact.

#### *Physiologic Protective Attributes of Skin*

Many of the skin's physiologic attributes participate in resisting the various insults that produce occupational skin diseases. Keratin, the outermost layer of the epidermis, is resistant to short-term immersion in water and to weak acids, but is vulnerable to the action of alkalis, detergents, strong soaps, solvents, and to prolonged immersion. The lipid emulsion on the surface of the keratin is composed of sebum, degradation products of keratin, and sweat. To a limited extent, the emulsion impedes the entrance of water and water-soluble chemicals and resists changes in the pH of the skin surface, but it is removed by solvents, soaps, detergents, emulsions, and prolonged immersion in water.

The transitional layer between the cornified and noncornified epidermis is an important barrier which prevents the loss of water from the skin and the entrance of most water-soluble chemicals. This subcorneal barrier layer

may be damaged by maceration, mechanical or chemical trauma, and certain internal diseases. Lipid-soluble and nonionizable (organic) substances are able to bypass the barrier layer and enter by way of the transfollicular pathway. Gases, except for carbon monoxide, are readily exchanged through the epidermis.

Protection against actinic stimuli is afforded by increased melanin production and a concomitant thickening of the keratin layer. An antimicrobial action is attributed to certain fatty acids in the surface lipid film, and the subcorneal barrier layer also offers some protection against microorganisms.

### *Primary Irritants*

Primary irritation causes most of the cases of contact dermatitis in industry. A primary irritant is an agent which will cause dermatitis by direct action on the normal skin at the site of contact if it is permitted to act in sufficient intensity or quantity for a sufficient length of time. Thus, the normal skin will almost always react to a primary irritant if the necessary conditions are present.

Irritants can act in several ways to injure the skin. Several examples have already been given, such as removal of lipid film, denaturation of keratin, and interference with the subcorneal barrier layer. Other examples of primary irritation include dehydration by inorganic acids, anhydrides, and alkalis; protein precipitation by heavy metal salts and tanning agents; and oxidation by bleaches, chlorine compounds, and per- salts.

### *Sensitizers*

Ordinarily, no more than 20 percent of contact dermatitis in industry is caused by allergens. However, certain of the strong sensitizers may affect many workers. Almost any chemical can be a sensitizer, but the mode of action usually differs from that exhibited by primary irritants. The cutaneous sensitizer, therefore, does not necessarily cause demonstrable cutaneous change on first contact, but may effect such specific changes in the skin so that after 5 to 7 days or more, further contact on the same or other parts of the body may cause dermatitis.

The difference between the irritant and the sensitizer, therefore, is generally a matter of time as well as mode of action. The irritant will usually act within a matter of minutes to a few hours, whereas the sensitizer requires at least 5 to 7 days because sensitization must build up during the period of incubation. Low-grade irritants such as soap may require prolonged or repeated contact before a dermatitis appears, and this prolonged reaction time may be confused with allergic incubation.

### *Secondary Effects*

After the cutaneous defenses have been broken down and a contact dermatitis is present, the oozing or fissured surface offers ideal conditions for the entrance and growth of bacteria. Thus, it is not unusual for a contact

dermatitis to be secondarily infected. Another secondary effect commonly seen in contact dermatitis is over-treatment by irritating or sensitizing medication.

### *Diagnosis*

Diagnosing an occupational dermatosis is generally contingent upon satisfying certain criteria. The disease should look like a contact dermatitis or one of the other clinical types of occupational dermatoses. It should be located on sites of exposure, and the time of appearance of the eruption as well as periods of remission and exacerbation should correlate with the history of work exposures. When the patient does not get well following complete withdrawal from the suspected contact agent, stimuli of nonoccupational origin should be reinvestigated. Multiple factors not associated with the occupation can perpetuate a chronic dermatosis.

Patch tests are helpful in differentiating between a primary irritation dermatitis and an allergic contact dermatitis. A nonirritating concentration of the suspected allergen is applied to the normal skin of a patient for 24 to 48 hours in an attempt to reproduce an eczematous dermatitis beneath the covering patch.

### *Classification of Lesions*

The clinical lesions seen among occupational exposures are multiple, varying from the mildest erythema to lesions of neoplastic nature. However, occupational skin disease can be classified as follows:

(1) Acute contact eczematous dermatitis characterized by erythema, edema, papules, vesicles, or bullae, crusts and finally desquamation. These effects are generally the result of contact with either a primary irritant or a sensitizer, or with both.

(2) Chronic contact eczematous dermatitis characterized by erythema, lichenification, and fissuring of the skin, usually resulting from contact with dehydrators, fat solvents, soaps, and detergents.

(3) Folliculitis and acneform types, including chloracne, characterized by plugged sebaceous follicles and suppurative lesions. Chloracne also shows numerous straw-colored cystic lesions. These forms of occupational dermatoses are usually caused by contact with oils, tars, waxes, and certain chlorinated hydrocarbons.

(4) Neoplastic (benign and malignant) types, characterized by keratoses, papillomata and epitheliomata of the exposed areas, usually caused by petroleum products, coal tar and certain derivatives, sunlight, and ionizing radiation.

(5) Pigmentary disturbances characterized by increase or decrease of pigment in the epidermis. Increased pigmentation can result from contact with

coal tar compounds, certain petroleum oils, vegetables, and fruits. Decreased pigmentation may result from trauma, dermatitis, or from contact with monobenzyl ether of hydroquinone.

### *Potential Occupational Exposures*

The following is a list of occupations each accompanied by certain agents frequently associated with that occupation and capable of producing a dermatosis. Additional agents for the occupations listed as well as additional occupations will be found in other sections, principally the one on chemical hazards.

#### ABRASIVE WHEEL MAKERS

abrasive dusts

resin glues

#### AGRICULTURAL WORKERS

*See Farmers*

#### AIRCRAFT WORKERS

bichromates

chlorinated solvents

chromates

chromic acid

cutting fluids

cyanides

glass fiber

hydraulic fluids

hydrofluoric acid

lubricants

nitric acid

oils

paints

plastics

resins

rubber

solvents

ultraviolet light

vibrating tools

X-rays

#### ANIMAL HANDLERS

bacteria

fungi

insecticides

parasites

pesticides

viruses

#### AUTOMOBILE WORKERS

asbestos

bichromates

brake fluids

cutting fluids

epoxy resins

gasoline

#### AUTOMOBILE WORKERS—continued

lead

lubricants

oils

paints

plastics

polyester resins

rubber

solvents

thinners

#### AVIATION MECHANICS

chlorinated solvents

fuels

hydraulic fluids

lubricants

oils and zinc chromate

used as aluminum-oxidation  
inhibitors

#### BAKERS

cinnamon

dough

dusts

flour improvers

fungi

heat

monilia

sugar

#### BARBERS AND HAIRDRESSERS

ammonium thioglycolate

bacteria

cosmetics

depilatories

detergents, synthetic

dyes

fungi

hair tonics

lacquer removers

nail lacquers

perfumes

soaps

## BARBERS AND HAIRDRESSERS—continued

ultraviolet light  
wave solutions

## BARREL WASHERS

bleaches  
deodorants  
detergents, synthetic  
soaps  
sodium carbonate  
solvents  
trisodium phosphate

## BARTENDERS

citrus fruits  
detergents, synthetic  
monilia  
soaps

## BASKET WEAVERS

essential oils  
fungi

## BATH ATTENDANTS

fungi  
linaments  
oils  
tonics  
ultraviolet light

## BATTERY MAKERS

benzol  
carbolic acid  
glass fiber  
mercury  
pitch  
sulfuric acid  
tar  
zinc chloride

## BLEACHERS

chlorine compounds  
chromium compounds  
hydrochloric acid  
hydrofluoric acid  
hydrogen peroxide  
nitric acid  
oxalic acid  
per-salts  
potassium hydroxide  
sodium hydroxide  
sodium silicate  
sulfur compounds

## BOOKBINDERS

formalin  
glues  
inks

## BOOKBINDERS—continued

oxalic acid  
shellac  
solvents

## BRICK MASONS

cement  
cold  
epoxy resins  
lime  
moisture  
sunlight

## BRIQUETTE MAKERS

coal tar pitch

## BRONZERS

acetone  
ammonia  
ammonium sulfide  
amyl acetate  
antimony sulfide  
arsenic  
arsine  
benzine  
benzol  
cyanides  
heat  
hydrochloric acid  
lacquers  
mercury  
methyl alcohol  
petroleum hydrocarbons  
phosphorus  
resins  
sodium hydroxide  
sulfur dioxide  
turpentine  
varnishes

## BROOM AND BRUSH MAKERS

bacteria  
bleaches  
colophony resin  
dust, vegetable  
dyes  
fungi  
glues  
parasites  
pitch  
plastics  
rubber  
shellac  
solvents  
tar

BROOM AND BRUSH MAKERS—continued	spices
varnish	
woods	
BUTCHERS	tartaric acid
antibiotics	CANNERS
bacteria	bacteria
detergents, synthetic	citrus oil
fungi	dyes
moisture	fruit acids
BUTTON MAKERS	fungi
bacteria	moisture
dusts	monilia
dyes	parasites
hydrogen peroxide	resins
plastics	salt
CABINET MAKERS AND CARPENTERS	vegetable juices
bleaches	CAP LOADERS, PERCUSSION
glues	mercury compounds
oils	CARPENTERS
rosin	<i>See</i> Cabinet makers and carpenters
shellac	CARPET MAKERS
solvents	alizarine
stains	aniline dyes
synthetic resins	anthrax bacillus
varnish	bleaches
woods	chlorine
<i>See also</i> Woodworkers	fungicides
CABLE WORKERS AND SPLICERS	glues
chlorodiphenyls	insecticides
chloronaphthalenes	loom oils
dyes	solvents
epoxy resins	CARROTTERS, FELT HAT
solvents	acids
CANDLE MAKERS	mercury compounds, if used
ammonium chloride	CARTRIDGE DIPPERS
ammonium phosphate	acids
ammonium sulfate	soaps
borax	CASE HARDENERS
boric acid	heat
chlorine	oils
chromates	sodium carbonate
hydrochloric acid	sodium cyanide
potassium nitrate	sodium dichromate
sodium hydroxide	sodium nitrite
stearic acid	CELLULOSE WORKERS
waxes	carbon disulfide
CANDY MAKERS	finishing oils
chocolate	CEMENTERS, RUBBER SHOE
citric acid	benzol
essential oils	carbon disulfide
pineapple juice	

## CEMENTERS, RUBBER SHOE—continued

coal tar products  
methyl alcohol  
naphtha

## CEMENT WORKERS

cement  
chromates  
cobalt  
epoxy resins  
lime  
moisture  
pitch  
resins

## CHEMICAL WORKERS

*See Chemical Hazards section*

## CHROME PLATERS

chromium compounds  
degreasing solvents  
sulfuric acid

## CLERKS

adhesives  
carbon paper  
copy paper  
duplicating fluid removers  
duplicating materials  
indelible pencils  
ink removers  
inks  
rubber  
solvents  
type cleaners  
typewriter ribbons

## CLOTH PREPARERS

acids  
alkalis  
amino resins  
detergents, synthetic  
dyes  
formaldehyde  
fungicides  
moisture  
potassium salts  
soaps  
sodium metasilicate  
sodium salts  
sodium silicate

## COAL TAR WORKERS

anthracene oil  
benzol  
coal tar  
creosote

## COAL TAR WORKERS—continued

cresol  
naphtha  
pitch  
sunlight

## COMPOSITORS

alkalis  
inks  
solvents

## CONSTRUCTION WORKERS

adhesives  
cement  
cold  
creosote  
gasoline  
glass fiber  
oils  
paints  
pitch  
solvents  
sunlight  
ultraviolet light

## COOKS

fruit acids  
heat  
moisture  
monilia  
spices  
sugar  
vegetable juices

## COTTON SIZERS

acids  
aluminum salts  
arsenic salts  
calcium salts  
carbolic acid  
dicyanodiamide formaldehyde  
fungicides  
magnesium salts  
melamine formaldehyde  
sodium hydroxide  
starch  
urea formaldehyde  
zinc chloride

## DAIRY WORKERS

antibiotics  
bacteria  
detergents, synthetic  
fungi  
mites  
viruses

DEGREASERS	DOCK WORKERS—continued
solvents	heat
DEMOLITION WORKERS	insects
bacteria	irritating or infected cargoes
chemicals	mites
cold	moisture
fungi	petroleum
moisture	sunlight
sunlight	tar
TNT	DRUGGISTS
ultraviolet light	acids
DENTISTS	alkalis
anesthetics, local	antibiotics
antibiotics	bleaching powder
disinfectants	detergents, synthetic
eugenol	drugs
ionizing radiation	iodoform
mercury amalgams	soaps
oil of clove	sodium salts
phosphoric acid	sugar
plastics	DRY CLEANERS
soaps	acetic acid
DETONATOR CLEANERS, FILLERS AND	ammonia
PACKERS	amyl acetate
mercury compounds	benzine
DISHWASHERS	carbon tetrachloride
bacteria	chlorobenzene
detergents, synthetic	dusts
grease	methanol
moisture	nitrobenzene
monilia	perchloroethylene
soaps	sizing compounds
water softeners	Stoddard solvent
DISINFECTANT MAKERS	trichloroethylene
carbolic acid	turpentine
chloride of lime	waterproofing compounds
chlorine	DYE MAKERS
cresol	acids
formaldehyde	alkalis
iodine	antimony compounds
mercury compounds	benzine
surfactants	calcium salts
zinc chloride	carbolic acid
DOCK WORKERS	coal tar products
bacteria	cresol
castor bean pomace	dextrins
chemicals	dye intermediates
cold	ferrocyanides
fumigants	formaldehyde
fungi	gums
grains	hydroquinone

## DYE MAKERS—continued

lead salts  
potassium chlorate

## DYERS

acids  
alkalis  
bleaches  
detergents, synthetic  
dyes  
mercurial salts  
moisture  
solvents  
zinc chloride

## ELECTRIC APPARATUS MAKERS

acids  
asbestos  
enamels  
epoxy resins  
ionizing radiation  
phenolic resins  
pitch  
rubber  
solder fluxes  
solvents  
synthetic waxes  
varnishes

## ELECTRICIANS

chlorinated diphenyls  
chlorinated naphthalenes  
electricity  
epoxy resins  
solder fluxes  
solvents  
waxes, synthetic

## ELECTROPLATERS

acids  
alkalis  
benzine  
chromic acid  
heat  
lime  
moisture  
nickel  
potassium cyanide  
soaps  
waxes, synthetic  
zinc chloride  
zinc cyanide

## EMBALMERS

bacteria  
carbolic acid

## EMBALMERS—continued

formaldehyde  
fungi  
ionizing radiation  
mercury  
oil of cinnamon  
oil of clove  
thymol  
zinc chloride

## ENAMELERS

acids  
alkalis  
arsenic  
chromium  
cobalt  
nickel

## ENGRAVERS

acids  
alkalis  
ferric chloride  
potassium cyanide  
solvents  
tropical woods

## ETCHERS

acids  
alkalis

## EXPLOSIVE WORKERS

ammonium salts  
mercury compounds  
nitroglycerin  
PETN  
picric acid  
tetryl  
TNT

## FARMERS

antibiotics  
bacteria  
cold  
detergents, synthetic  
feeds  
fertilizers  
fruits  
fungi  
heat  
lubricants  
oils  
parasites  
pesticides  
poison ivy  
poison oak  
poison sumac

FARMERS—continued	FOOD PRESERVERS—continued
ragweed	vinegar
solvents	waxes
sunlight	
vegetables	
FELT HAT MAKERS	FOUNDRY WORKERS
acids	acids
bacteria	heat
dyes	lime
Glauber's salt	resins
hydrogen peroxide	ultraviolet light
mercuric nitrate, if used	
sodium carbonate	
FERTILIZER MAKERS	FURNACE WORKERS
acids	heat
ammonium compounds	ultraviolet light
calcium cyanamide	
castor bean pomace	
fluorides	
lime	
manure	
nitrates	
pesticides	
phosphates	
potassium salts	
FISH DRESSERS	FURNITURE POLISHERS
bacteria	alkalis
brine	benzine
cold	methyl alcohol
moisture	naphtha
redfeed	pyridine
sunlight	rosin
trauma	soaps
	turpentine
	waxes
FLAX WORKERS	FUR PROCESSORS
brine	acids
lime	alkalis
	alum
FLOUR MILL WORKERS	bacteria
dust	bleaches
fungi	chromates
parasites	dyes
pesticides	formaldehyde
	fungi
FOOD PRESERVERS	lime
bleaches	oils
brine	salt
ionizing radiation	
moisture	
monilia	
resins	
spices	
sugar	
	GALVANIZERS
	acids
	ammonium chloride
	zinc chloride
	GARAGE WORKERS
	antifreeze solutions
	detergents, synthetic
	epoxy resins
	gasoline
	gasoline additives
	glass fiber
	greases
	moisture
	oils
	paint removers
	paints

**GARAGE WORKERS**—continued

polyester resins  
solvents

**GARDENERS**

fertilizers  
fungi  
fungicides  
herbicides  
insecticides  
insects  
plants  
poison ivy  
poison oak  
sunlight

**GAS MANTLE MAKERS**

thorium compounds

**GLASS WORKERS**

arsenic  
borax  
boric acid  
glass fiber  
glass wool  
heat  
hydrofluoric acid  
lead compounds  
lime  
metallic oxides  
petroleum oils  
resins  
soda ash  
ultraviolet light

**HAIRDRESSERS**

*See* Barbers and hairdressers

**HIGHWAY WORKERS**

*See* Road workers

**HISTOLOGY TECHNICIANS**

alcohol  
benzol  
formaldehyde  
mercury bichloride  
osmium tetroxide  
potassium dichromate  
stains  
toluene  
waxes  
xylene

**INK MAKERS**

anti-skinning agents  
chromates  
cobalt compounds

**INK MAKERS**—continued

detergents, synthetic  
dyes  
ethyl acetate  
ethyl alcohol  
mercuric chloride  
soaps  
solvents  
turpentine  
varnish

**INSECTICIDE MAKERS**

aldrin  
allethrin  
arsenic trioxide  
calcium arsenate  
chlordane  
DDT  
dieldrin  
lindane  
malathion  
methoxychlor  
parathion  
piperonyl compounds  
pyrethrin  
strobane  
*See also* Pesticides section

**JANITORS**

bacteria  
detergents, synthetic  
disinfectants  
polishes  
soaps  
solvents  
waxes

**JEWELERS**

acids  
chromates  
cyanides  
mercury  
mercury solvents  
nickel  
solder fluxes

**LABORATORY WORKERS, CHEMICAL**

acids  
alkalis  
chromates  
detergents, synthetic  
moisture  
organic solvents  
soaps

## LAUNDRY WORKERS

alkalis  
bactericides  
bleaches  
chemical dusts  
detergents, synthetic  
heat  
soaps

## LINOLEUM MAKERS

asphalt  
dyes  
oils  
pigments  
resins

## LONGSHOREMEN

*See* Dock workers

## MACHINISTS

chlorinated cutting oils  
chromates  
cutting fluids  
germicides  
lubricating oils  
rust inhibitors  
solvents

## MASONS

*See* Brick masons

## MATCH FACTORY WORKERS

ammonium phosphate  
chromates  
dextrins  
dyes  
formaldehyde  
glues  
gums  
phosphorus sesquisulfide  
potassium chlorate  
red phosphorus  
waxes

## MEAT PACKERS

*See* Butchers

## MECHANICS

*See* Aviation mechanics, and Garage workers

## MERCERIZERS

acids  
alkalis

## METAL POLISHERS

abrasives  
acids  
alkalis

## METAL POLISHERS—continued

ammonia  
naphtha  
pine oil  
potassium cyanide  
soluble oils  
soaps  
solvents  
triethanolamine  
waxes

## MIRROR MAKERS

ammonia  
cyanides  
formaldehyde  
lacquers  
silver nitrate  
solvents  
tartaric acid  
varnishes

## MORDANTERS

acids  
alkalis  
aluminum salts  
antimony compounds  
arsenates  
chromates  
copper salts  
iron salts  
lead salts  
phosphates  
silicates  
tin salts  
zinc chloride

## NICKEL PLATERS

detergents, synthetic  
heat  
moisture  
nickel sulfate  
zinc chloride

## NITROGLYCERIN MAKERS

ethylene glycol dinitrate  
nitric acid  
nitroglycerin  
sodium carbonate  
sulfuric acid

## NURSES

anesthetics, local  
antibiotics  
antiseptics  
bacteria  
detergents, synthetic

**NURSES—continued**

disinfectants  
drugs  
fungi  
ionizing radiation  
moisture  
soaps  
tranquilizers  
viruses

**OIL FIELD WORKERS**

alkalis  
brine  
crude petroleum  
ionizing radiation  
lubricating oils  
sunlight

**OPTICAL WORKERS**

alkalis  
grinding fluids  
oils  
turpentine

**PACKING-HOUSE WORKERS**

*See* Slaughter- and packing-house workers

**PAINTERS**

acetone  
acids  
alkalis  
benzene  
chromates  
drying agents  
paint removers  
paints  
pigments  
resins  
solvents  
sunlight  
thinners  
turpentine

**PAINT MAKERS**

anti-mildew agents  
chromates  
coal tar distillates  
drying agents  
fish oils  
latex  
oil, vegetable  
petroleum solvents  
pigments  
resins  
thinners

**PAINT MAKERS—continued**

turpentine  
zinc chloride

**PAPER BOX MAKERS**

dyes  
glues  
plastics  
resins  
waxes

**PAPER MAKERS**

alkalis  
aluminum sulfate  
calcium bisulfite  
calcium chloride  
chromates  
glues  
heat  
moisture  
resins  
rosin  
sodium hydroxide  
sodium sulfate  
sodium sulfide  
sulfur dioxide

**PARAFFIN WORKERS**

paraffin  
paraffin distillates  
solvents

**PARCHMENT MAKERS**

zinc chloride

**PENCIL MAKERS**

aniline dyes  
chromium pigments  
glues  
gums  
lacquer  
lacquer thinners  
methyl violet  
pyridine  
red cedar wood  
resins  
solvents  
waxes

**PETROLEUM REFINERY WORKERS**

acids  
alkalis  
aluminum chloride  
arsenic  
gas oil  
gasoline  
hydrofluoric acid

## PETROLEUM REFINERY WORKERS—con.

kerosine  
paraffin  
paraffin distillates  
petroleum  
petroleum solvents  
tar  
waxes

## PHOTOENGRAVERS

ammonium bichromate  
etching acids  
inks  
photographic developers  
solvents  
ultraviolet light

## PHOTOGRAPHERS

acids  
alkalis  
chromates  
hydroquinone  
methyl-para-aminophenol sulfate  
para-aminophenol  
paraformaldehyde  
paraphenylenediamines  
photographic developers  
pyrogallic acid  
sodium hypochlorite  
sodium sulfide  
turpentine

## PHYSICIANS

anesthetics, local  
antibiotics  
antiseptics  
bacteria  
detergents, synthetic  
drugs  
fungi  
ionizing radiation  
rubber gloves  
soaps  
tranquilizers  
viruses

## PITCH WORKERS

heat  
pitch  
solvents  
sunlight  
tar

## PLASTERERS

lime  
moisture

## PLASTIC AND RESIN MAKERS

*See* Plastics and Synthetic Resins section

## PLUMBERS

cement  
cold  
hydrochloric acid  
parasites  
solvents  
tar  
zinc chloride

## PRINTERS

alkalis  
aniline  
chromates  
glues  
gum arabic  
inks  
solvents

## RAILROAD SHOP WORKERS

alkalis  
antiseptics  
chromates  
cutting fluids  
detergents, synthetic  
dichlorobenzene  
diesel fuel oil  
greases  
insecticides  
lacquers  
lubricating oils  
magnaflux  
paint  
paint strippers  
paint thinners  
solvents  
ultraviolet light

## RAILROAD TRACK WORKERS

cold  
creosote  
fungicides  
herbicides  
pitch  
poison ivy  
poison oak  
poison sumac  
ragweed  
sunlight  
tar

## RAYON WORKERS

acetic anhydride  
acids  
alkalis  
ammonium sulfide  
bleaches  
calcium bisulfite  
carbon disulfide  
coning oils  
sodium cyanide  
sodium sulfide  
sodium sulfite  
solvents

## REFRIGERATION WORKERS

ammonia  
brine  
cold  
dry ice  
ethyl bromide  
ethyl chloride  
glass fiber  
methyl chloride  
sulfur dioxide

## ROAD WORKERS

asphalt  
cement  
cold  
epoxy resins  
herbicides  
paint  
parasites  
pitch  
poison ivy  
poison oak  
poison sumac  
ragweed  
sunlight  
tar

## ROCKET FUEL HANDLERS

aniline  
boron hydrides  
chlorine trifluoride  
dimethylhydrazine  
ethyl oxide  
fuming nitric acid  
gasoline  
hydrazine  
hydrogen fluoride  
hydrogen peroxide  
kerosine  
liquid oxygen

## ROPE MAKERS

alkalis  
bleaches  
dusts  
dyes  
oils  
pitch  
soaps  
tar

## RUBBER WORKERS

accelerators  
acids  
activators  
adhesive removers  
alkalis  
antioxidants  
benzol  
chloroprene dimers  
chromium pigments  
curing agents  
formaldehyde  
heat  
oils  
plasticizers  
resins  
retarders  
soaps  
solvents  
tar  
turpentine  
zinc chloride

## SHIPYARD WORKERS

asbestos  
chlorinated diphenyls  
chlorinated naphthalenes  
chromates  
cold  
fungicides  
glass fiber  
paint removers  
paints  
paint thinners  
resins  
solvents  
tar  
ultraviolet light  
wood preservatives

## SHOEMAKERS (MANUFACTURERS)

adhesives  
ammonia  
amyl acetate  
amyl alcohol

## SHOEMAKERS (MANUFACTURERS)—con.

aniline dyes  
benzine  
benzol  
coal tar products  
methyl alcohol  
naphtha  
plastics  
rubber  
shoe polishes  
waxes

## SILK PROCESSORS

acids  
alkalis  
dyes  
xylene

## SLAUGHTER- AND PACKING-HOUSE WORKERS

antibiotics  
bacteria  
brine  
cold  
detergents, synthetic  
enzymes  
fungi  
parasites  
spices

## SOAP MAKERS

alkalis  
bacteriostats  
detergents, synthetic  
oil, vegetable  
perfumes  
sodium silicate

## SOLDERERS

acids  
cyanides  
fluxes  
heat  
hydrazine salts  
rosin  
zinc chloride

## STEVEDORES

*See* Dock workers

## STOCKYARD WORKERS

bacteria  
fungi  
insecticides  
parasites

## STONE WORKERS

cement

## STONE WORKERS—continued

dusts  
lime  
vibrating tools

## SUGAR REFINERS

acid  
burlap  
heat  
jute  
lime  
monilia  
sugar

## TANNERY WORKERS

acetic acid  
acids  
alum  
ammonium chloride  
arsenic salts  
bacteria  
benzol  
brine  
calcium hydrosulfide  
chromates  
dimethylamine  
dyes, mineral  
dyes, vegetable  
formaldehyde  
lime  
oils  
pancreatic extract  
sodium hydroxide  
sodium sulfide  
solvents  
tannin

## TAR WORKERS

heat  
pitch  
solvents  
sunlight  
tar

## TAXIDERMISTS

anthrax bacillus  
arsenic  
bacteria  
calcined alum  
fungi  
mercuric chloride  
parasites  
solvents  
tannin  
zinc chloride

## TEMPERERS

oils  
sodium carbonate  
sodium cyanide  
sodium dichromate  
sodium nitrite

## TINNERS

pitch  
sunlight  
zinc chloride

## TOBACCO WORKERS

dust, vegetable  
glues  
glycerine  
insecticides  
oil, vegetable

## TYPISTS

*See Clerks*

## UNDERTAKERS

*See Embalmers*

## UPHOLSTERERS

bacteria  
fungi  
glues  
lacquer  
lacquer solvents  
methyl alcohol  
parasites

## VETERINARIANS

anesthetics, local  
antibiotics  
bacteria  
carbon disulfide  
drugs  
fungi  
mercuric chloride  
parasites  
pesticides  
viruses

## WATCHMAKERS

acids  
chromates  
nickel salts  
potassium cyanide  
solvents

## WATERPROOFERS

alum  
Japan wax  
melamine formaldehyde resins  
oils  
paraffin  
pitch  
rubber  
solvents  
waxes

## WELDERS

fluxes  
heat  
ultraviolet light

## WIRE DRAWERS

drawing oils  
lime  
soaps  
sulfuric acid

## WOOD PRESERVERS

chlorophenols  
chromates  
copper compounds  
creosote  
cresols  
mercuric chloride  
phenyl mercuric compounds  
resins  
tar  
zinc chloride  
zinc sulfate

## WOODWORKERS

acid bleaches  
amino resin glues  
fillers  
formaldehyde  
glues  
lacquers  
mercuric chloride  
oil stains  
paints  
phenolic resin glues  
rosin  
solvents  
varnishes  
woods  
*See also Cabinet makers and carpenters*

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## • section V

### PNEUMOCONIOSES

THOMAS H. MILBY, M.D.

The word pneumoconiosis literally means dust retained in the lung, with no implication as to whether disease is or is not present. In more common usage, however, the word has become a general term for any of the dust diseases of the lung and is used here with that meaning.

The development of a pneumoconiosis depends upon a number of factors related to the worker and to the dust. Worker factors include duration of exposure to the dust and susceptibility; factors related to the dust include its chemical composition, particle size, and concentration.

There have been suggested many different classifications for the pneumoconioses. They have been classified according to etiology, pathology, and physiology. The offending dusts themselves have been classified according to origin, and chemical, physical, and noxious properties. Notwithstanding the numerous efforts to systematize the information available concerning the pneumoconioses, no single classification has been widely accepted.

In this presentation, there has been neither an attempt to classify these dust diseases nor to discuss all of those described in the available medical literature. Included are only the pneumoconioses which are best understood, most commonly seen, and most widely accepted as definite clinical entities. These include:

(1) Silicosis	(6) Talcosis
(2) Coal Workers'	(7) Pulmonary Siderosis
Pneumoconiosis	(8) Byssinosis
(3) Asbestosis	(9) Bagassosis
(4) Diatomite Pneumoconiosis	(10) Farmer's Lung
(5) Shaver's Disease	

The following dusts and their effects when inhaled have not been included even though they have been reported as having caused pneumoconiosis: mica, kaolin, feldspar, cement, gypsum, fluorspar, sepiolite, sulfur, jute, moura seed, and grain.

Since the discussions that follow apply generally to dusts containing a single pathogenic substance, it should be recognized that where exposure occurs to dusts containing several harmful substances, the entire response may be markedly altered and result in the production of many bizarre findings.

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### (1) Silicosis

*grinders' rot, miners' consumption, miners' phthisis, potters' asthma, stone masons' phthisis*

Silicosis is a pneumoconiosis caused by the inhalation of finely divided silicon dioxide (silica) in the free state, which may be in a crystalline form such as in quartz, cristobalite and tridymite, or in a noncrystalline or amorphous form such as in opal. It has been shown that the crystal structure of pure silica has an important influence upon tissue reaction. Thus, in the production of a fibrous tissue response, tridymite is intensely fibrogenic, cristobalite and quartz, are somewhat less fibrogenic, and finally, amorphous silica is only slightly fibrogenic.

Silica in the nonfree or combined state, namely a silicate, refers to silica in chemical combination. Thus, the feldspars are aluminum silicates with potassium, sodium, calcium, or barium. Other silicates include kaolin, mica, serpentine, shale, slate, and talc. A pneumoconiosis associated with the inhalation of the dust of a silicate is termed a silicatosis.

Silica and silicates, composing almost entirely the crust of the earth, constitute the major portion of all rocks and their products such as soils, sands, and clays.

It is generally accepted that the size of the offending silica particle is of extreme importance in determining the degree of tissue reaction that will occur in the lung following the inhalation of siliceous dust. The size of the particle directly influences the concentration of particles that may be suspended in the air; it also determines the depth to which these particles penetrate into the lung and in what amounts they may become deposited and retained. While experimental silicosis has been produced with particles as large as 8 to 10 microns in diameter, it has been reported that the optimum size for alveolar retention of silica dust is about 1 micron. Recent evidence suggests, however, that particles below 1 micron in size may be the most dangerous since they penetrate deep into the alveolar spaces and are deposited there in very high concentrations. The lower limit of particle size which will produce a fibrogenic reaction is unknown, but may be close to 0.1 micron.

Silicosis may be either of an acute or of a chronic nature. The former is referred to as rapidly-developing silicosis rather than as acute silicosis.

The etiology, symptomatology, and pathology of rapidly-developing silicosis are not well understood. The disease has been most often reported in manufacturers and packers of abrasive soap powders, in sand-blasters working in enclosed tanks, and in high-power drillers of tunnel rock. It was suggested in 1939 at the Fourth Saranac Laboratory Symposium that one or more factors may have important etiologic significance. Such factors are exposure to very finely divided crystalline silica dust; exposure to massive amounts of free crystalline silica; synergistic action of other ions; differences in individual susceptibility; and presence of concomitant infection, especially tuberculosis.

The time of exposure to silica dust was relatively short in the reported cases of rapidly-developing silicosis, varying from 8 to 18 months from the time of the first exposure to the time of the onset of symptoms. After development of symptoms, the survival time is likely to be very short. The clinical picture of this type of silicosis is characterized by pulmonary insufficiency, with dyspnea, tachypnea, and cyanosis leading to the development of cor pulmonale. Many cases have been complicated by pulmonary tuberculosis. The chest roentgenogram in rapidly-developing silicosis shows diffuse fibrosis with no visible typical nodulation. Roentgenographic evidence of pulmonary tuberculosis is often present.

Chronic pulmonary silicosis, the type usually encountered in industry, is produced, as a rule, only after years of silica dust inhalation. The disease is reported to occur most commonly in the mining industries but is also seen in numerous other industries such as potteries, foundries, stone cutting and finishing, tile and clay producing, and glass manufacturing.

Although silicosis may be identified in a relatively early stage with the aid of a satisfactory chest roentgenogram, the uncomplicated disease may progress to an advanced stage while producing only symptoms of moderate dyspnea. The shortness of breath is noted first on moderate exertion, but as the disease progresses, the dyspnea occurs with less and less exertion.

Clinically, silicosis may follow one of several courses. The simple, uncomplicated form, frequently called simple discrete nodular silicosis, often does not progress beyond the stage where the nodules comprise a relatively small amount of the total lung tissue. This form of silicosis may present itself symptomatically only as a slowly increasing, non-disabling, exertional dyspnea. The chest roentgenogram usually reveals, in this form of the disease, uniformly distributed, discrete densities up to 10 mm in diameter. There is also very often seen enlargement of the shadows cast by the tracheo-bronchial lymph nodes.

In some silicotic patients, there is seen to develop in the upper portions of both lungs, large irregular masses of dense fibrous tissue. When these conglomerate masses appear on the chest roentgenogram, the disease may be categorized as conglomerate silicosis. In this form of the disease, the presence of advanced fibrosis and diffuse, obstructive emphysema may lead to severe respiratory crippling due to a decrease in the maximum breathing capacity and an increase in the residual lung volume. At this stage, the clinical symptoms, in addition to dyspnea on exertion, may include a productive cough, chest pain, and marked weakness. Cor pulmonale, probably caused by the increase in pressure required to force blood through a damaged pulmonary capillary bed, is a late and frequently fatal complication.

Tuberculosis is considered a common complication of silicosis. This combination is frequently manifested by the appearance of coalescent or conglomerate shadows on a chest roentgenogram which previously had demonstrated only shadows suggestive of simple discrete nodular silicosis.

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## (2) Coal Workers' Pneumoconiosis

The dust to which a coal worker may be exposed is complex in nature. Besides the dust arising from the disintegration of coal, which itself is always intimately associated with other minerals, siliceous dusts of various types are derived from the rock strata above and below the coal seam. The relative importance of the coal and the mineral dust in the production of coal workers' pneumoconiosis is a question that is continually debated. At present, however, there is growing agreement as to the terminology, namely, coal workers' pneumoconiosis, applicable to the pathologic condition of the lung resulting from exposure to coal dust.

The term anthracosis refers to a blackish pigmentation of the lungs caused by deposition of carbon particles and may be observed with no evident pathologic change at autopsy. The condition is frequently observed in the lungs of city dwellers who have had no industrial exposure. Since no apparent disease or disability is associated with this deposition of pigment, anthracosis will not be considered further here.

The term anthracosilicosis generally means a modified form of classical silicosis resulting from prolonged exposure to coal dust and to rock dust containing significant amounts of free silica. The pathologic condition observed in this form of pulmonary disease is essentially the deposition of coal dust in the lungs accompanied by extensive fibrosis, both diffuse and nodular, with associated functional changes.

The term coal workers' pneumoconiosis, as described by a number of authors, is a different disease, however, since there is little or no evidence of classical silicosis or of significant silica exposure. The disease, apparently caused by coal dust itself, is an established entity and is pathologically distinct from silicosis. In the simple or uncomplicated form of coal workers' pneumoconiosis, the lungs contain large quantities of coal dust which is aggregated into foci surrounding the respiratory bronchioles, frequently causing them to dilate, a condition known as focal emphysema. The fibrosis produced is strikingly sparse, the coal dust being held in a fine mesh of reticulin fibrils, stellate in appearance, and contrasting markedly with the rounded, whorled nodule of silicosis.

The advanced or complicated form of coal workers' pneumoconiosis starts within a few coal foci as a collagen fibrosis and subsequently enlarges and coalesces to form a dense mass of fibrous tissue. This fibrosis may occupy much of a lobe or even a whole lung, and is thought usually to be due to tuberculosis superimposed upon a lung heavily laden with coal dust. Because of the nature of this condition, it is often referred to as progressive massive fibrosis (PMF) and carries with it the implications of a grave prognosis, death frequently resulting from tuberculosis or

pulmonary insufficiency or from cor pulmonale secondary to obliteration of the pulmonary vascular bed by fibrous tissue invasion.

The roentgenographic characteristics of simple coal workers' pneumoconiosis include discrete opacities up to 10 mm in diameter, which may be arranged in groups or spread diffusely throughout the lung fields.

In the complicated form of the disease, the earliest roentgenographic evidence of PMF is the presence, usually on a background of simple pneumoconiosis, of larger, less well defined opacities, often resembling reinfection-type tuberculosis in both position and appearance. These large shadows tend to increase in size and to coalesce. They later may contract with resultant severe distortion of the lung architecture.

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### (3) Asbestosis

Asbestos is a general term used to describe several fibrous mineral silicates which differ in their chemical composition and physical properties. The most important types of asbestos are chrysotile, a simple magnesium silicate; amosite and anthophyllite, which are complex magnesium iron silicates; and crocidolite, a complex sodium iron silicate. About 95 percent of the world's asbestos production is derived from chrysotile. Deposits of this mineral are found in many countries, but the largest mines are located in Canada.

Prolonged inhalation of asbestos fibers between 20 and 50 microns long may result in the production of a typical pulmonary fibrosis which may be accompanied by severe respiratory disability. On the basis of experimental studies of asbestosis, it was reported in 1951 that this fibrosis is due to the mechanical action of the asbestos fiber. The fibers, upon being deposited in the terminal bronchioles, initiate a tissue response which results in the coating of the fiber with the ultimate production of what is known as the asbestos or asbestosis body. This response appears to be a defense mechanism of the lung. If large quantities of the fibers are inhaled over a prolonged period of time, characteristically 10 to 20 years, the tissue reaction progresses until a generalized, diffuse fibrosis becomes evident. This fibrosis is seen first in the lower lobes of the lungs but eventually, if exposure continues, appears in the other lobes as well. Respiratory insufficiency and cardiac failure may supervene. It is of considerable interest and significance that asbestos fibers smaller than about 20 microns in length are thought to be incapable of initiating a fibrogenic response.

The roentgenogram of the chest with pulmonary fibrosis resulting from prolonged inhalation of asbestos fibers discloses a typical pattern. In the early or first stages of the disease, the shadows are fine, diffuse and homogeneous and appear characteristically at the base of both lungs. The typical nodular pattern of silicosis is not seen in asbestosis; rather, the affected lung fields present a ground glass appearance.

In moderately advanced or second-stage asbestosis, the infiltration is more in evidence but remains generally confined to the lower lobes. The heart borders may become indistinct or shaggy, a condition which has been referred to as porcupine heart.

In far advanced or third-stage asbestosis, the infiltrate can be seen throughout the middle and upper lung fields; however, the apices generally remain clear. There is almost complete obliteration of the cardiac outline, the domes of the diaphragm and the costophrenic sulci.

It should be emphasized that the chest roentgenogram cannot accurately be used to estimate the presence or extent of impaired pulmonary function or disability in lung diseases in general, and in asbestosis in particular, since many individuals with radiographic evidence of third-stage asbestosis have been able to carry on their usual work and live fairly comfortable lives for several years. On the other hand, definite disability due to asbestosis has rarely been reported in the absence of a typical radiographic pattern.

There is no typical clinical picture for asbestosis. The disease is insidious in its onset and is slowly progressive so long as inhalation of the fiber continues. There is a gradual increase in cough and expectoration, anorexia, and weight loss, all combined with slowly increasing dyspnea. Cyanosis and clubbing of the fingers are rare findings. When an acute pneumonitis

develops in the presence of established asbestosis with fibrosis, recovery is often delayed because healing is slow and relapses are frequent.

The primary functional abnormality in pulmonary asbestosis is one of impaired oxygen transfer across the alveolar membrane rather than impairment of ventilatory capacity. This condition is referred to as an alveolar-capillary block.

Conflicting opinions and differences in reports make it difficult to confirm or deny conclusively a causal relationship between asbestosis and cancer of the lung or extrapulmonary tissues. However, there is increasing evidence to suggest that such a relationship exists.

With regard to the relationship between asbestosis and tuberculosis, it is fairly well established that asbestosis does not predispose to the development of tuberculosis, nor does it aggravate an apparently healed lesion.

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### (4) Diatomite Pneumoconiosis

Diatomite, frequently designated diatomaceous earth, diatomaceous silica, or kieselguhr, is composed of the siliceous skeletons of microscopic, unicellular, aquatic plants known as diatoms. Because of its remarkable properties, this nonmetallic mineral has found many industrial uses, such as in filters, insulators, absorbents, and polishes.

Crude diatomite is essentially amorphous silica and contains less than 5 percent of quartz and only traces of cristobalite and tridymite; however, after being processed by high-temperature calcining, the cristobalite content

may be as high as 60 percent. The particle size of finished diatomite powder products is predominantly under 10 microns.

As with most pneumoconiosis-causing dusts, the longer the exposure to diatomite dust, the more is the chance of developing demonstrable lung changes; however, it has been shown that exposure to this dust for as little as 1 to 3 years may produce definite roentgenographic evidence of pneumoconiosis. In addition, the extent and severity of diatomite pneumoconiosis correlate with the cristobalite content of the dust involved.

Radiographic changes resulting from exposure to diatomite dust can roughly be divided into two groups: (1) changes of a linear-nodular type, and (2) changes resulting in the production of coalescent opacities, usually superimposed on definite linear-nodular changes.

In this type of pneumoconiosis, pulmonary signs and respiratory symptoms correlate poorly with roentgenographic changes, except where massive confluent lesions are present, in which case pulmonary disability may be extreme.

When tuberculosis is superimposed on diatomaceous earth pneumoconiosis, the infection often pursues a benign course until cavitation supervenes; then the course is frequently one of slow deterioration despite modern treatment including collapse procedures and chemotherapy.

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## (5) Shaver's Disease

*corundum fume fibrosis, bauxite fume fibrosis*

As reported in the American literature, Shaver's disease is a pneumoconiosis of occupational origin, resulting from the inhalation of fume emitted by electric furnaces used in the production of corundum. This fume is rich in alumina and silica, both of which are in the free state and are largely amorphous in structure. The fume is further characterized by its small particle size, generally smaller than 0.5 micron and extending down to about 0.02 micron. Although the noxious agent or agents within this fume have not been identified, both silica fume and finely divided aluminum are thought to be capable of causing lung damage if inhaled in significant amounts.

In contrast to classical silicosis, Shaver's disease may develop in a remarkably short time, the period between first exposure and onset of symptoms being as brief as 24 months in some cases.

The most outstanding symptom of this disease is shortness of breath, usually mild in the early stages of illness but worsening as the disease progresses. Sudden attacks of extreme breathlessness are not uncommon and may indicate the occurrence of spontaneous pneumothorax, a condition seen with disturbing frequency among those afflicted with this disease. Additional signs and symptoms include cough productive of frothy white sputum, chest tightness and pain, weakness and fatigue.

The chest roentgenogram characteristically reveals bilateral granular haziness, widened mediastinum, heavy fibrotic strands, distortion and elevation of the diaphragm, and radiographic evidence suggestive of emphysematous bullae.

There is no evidence to suggest that this disease predisposes to pulmonary tuberculosis.

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### (6) Talcosis

Pure talc is a hydrated magnesium silicate, similar in chemical composition to asbestos. It is a flaky mineral, but also occurs in a fibrous state. When crushed, it forms a smooth bland powder which is used for a wide variety of purposes. The word talc, as used in industry, usually refers to a product which meets certain physical requirements rather than to a substance of definite chemical composition. Commercial talc varies markedly in its composition, and the mineral talc itself is usually only a minor component present in combination with other minerals such as dolomite, tremolite, magnetite, serpentine, mica, and anthophyllite. Varying amounts of free silica may also be present.

For practical purposes, then, the word talc as used here will refer to a mixture of minerals rather than to the specific mineral talc, which is hydrated magnesium silicate.

Numerous investigators have shown that prolonged inhalation of talc will result in the production of significant lung damage *even though there is little or no free silica present*. Histopathologic examination of lung sections usually reveals the presence of mild to moderate peribronchial and perivascular fibrosis with dilatation of many small bronchi and bronchioles. In more advanced cases the fibrosis may be extensive. Roentgenographic evidence of emphysematous bullae and fibrosis is usually demonstrable.

Some reports indicate that tremolite may be the main pathogenic agent in producing this characteristic talc lung lesion. The similarities between the histopathologic changes present in the talc lesion and those seen in the asbestos-produced lesion have been pointed out and are of considerable interest in view of the fact that tremolite is recognized as an asbestiform mineral.

A striking feature very frequently noted upon histologic examination of affected lung tissue sections is the presence of many brilliantly birefringent, needle-shaped particles in the areas of fibrosis. X-ray diffraction studies have indicated that these particles are talc. Another commonly reported finding is the presence of asbestos-like bodies embedded in the fibrous tissue. These structures have been most frequently seen in specimens of lungs which have been found to contain appreciable quantities of tremolite. They are less commonly seen in specimens which contain only small amounts of this mineral.

When the characteristic talc lesion is modified by significant amounts of free crystalline silica in the inhaled dust, the entire clinical, pathologic and roentgenographic picture may be greatly changed. There may be a greater tendency toward the formation of massive lesions, fibrosis may be more intense, and damage to the pulmonary vascular bed may be extreme. True classical silicotic nodules are uncommon in such cases.

Potential occupational exposures include cosmetic workers, paint makers, paper makers, pottery makers, rubber cable coaters, rubber tire makers, talc millers, talc miners, and talcum powder makers.

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### (7) Pulmonary Siderosis

Siderosis is a benign pneumoconiosis resulting from the deposition of inert iron dust in the lung. In general there is neither fibrosis nor emphysema associated with this condition unless, as often occurs, there is concomitant exposure to silica dust. Siderosis does not result in the production of disability nor does it show any predisposition to pulmonary tuberculosis or lung cancer.

The chest roentgenogram in siderosis closely resembles the picture seen in uncomplicated silicosis. There may be, in both conditions, discrete nodular densities evenly distributed throughout the lung fields. In siderosis when considered by itself, there is no emphysema and there is very little tendency toward the formation of the conglomerate masses which are often seen in silicosis.

The differential diagnosis between siderosis and silicosis is difficult, especially since they may occur together. The diagnosis can usually be made, however, on the basis of medical and occupational histories, physical examination, chest roentgenograms, pulmonary function studies, and an appraisal of the work environment.

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VORWALD, A. J.; PRATT, P. C.; DURKAN, T. M.; DELAHANT, A. B., AND BAILEY, D. A.: Siderosis, a benign pneumoconiosis due to the inhalation of iron dust. 2, An experimental study of the pulmonary reaction following inhalation of dust generated by foundry cleaning room operations. *Indust. Med. & Surg.* 19: 170, 1950.

### (8) Byssinosis

Byssinosis occurs in individuals who have experienced prolonged exposure to heavy air concentrations of cotton dust. Flax dust has also been incriminated. The exact mode of action of the cotton dust is unknown, but one or more of the following factors may be important in the pathogenesis of the disease: (1) Toxic action of microorganisms adherent to the inhaled fibers, (2) mechanical irritation from the fibers, and (3) allergic stimulation by the inhaled cotton fibers or adherent materials. There is no good evidence to suggest that pathogenic invasion by microorganisms plays a significant role in the etiology of byssinosis.

The earliest manifestations of byssinosis may become noticeable after several years of exposure to cotton dust. The worker at first develops slight dyspnea and tightness of the chest on reporting to work on Monday morn-

ings or on days immediately following holidays or absences. He usually recovers completely by the next day. During this early phase of the disease permanent removal from exposure to cotton dust generally results in permanent cessation of symptoms.

If the worker continues to be exposed to the dust, he may go for years without noticing a worsening of his Monday morning symptoms. In some cases, however, continued exposure to cotton dust over many years is attended by a slowly progressing increase in both duration and severity of symptoms as well as by the onset of cough, frequently productive in nature. Even if further exposure to cotton dust is terminated, workers who have progressed to this phase of byssinosis may experience a permanent reduction in exercise tolerance.

In the most advanced stages of the disease, cough, chest tightness, and dyspnea may be so severe that the worker is forced to leave the cotton industry. Although some relief may be experienced when exposure to cotton dust ceases, chronic bronchitis and generalized, nonspecific pulmonary emphysema usually remain to cause permanent disability. Cor pulmonale may develop in severe cases.

The diagnosis of byssinosis is based on (1) a history of exposure to cotton dust over a period of years and (2) the occurrence of dyspnea and chest tightness which appear when the cotton worker reports to work on Monday morning or on days following holidays or other absences.

Differentiation between byssinosis and unassociated chronic bronchitis is based on observation that patients with chronic bronchitis may experience chest tightness when exposed to any excessively dusty atmosphere and the worker with early byssinosis is affected only by cotton dust and is worse on returning to work after several days of absence, typically on Monday.

Pulmonary function studies performed on workers exposed to cotton dust have been reported to show significant decreases in ventilatory capacity as measured by tests of air way resistance and indirect maximum breathing capacity. These studies have been especially revealing when performed at the beginning and again at the end of the same work day. Inhalation of cotton dust does not initiate a fibrogenic response. There is no characteristic pattern identifiable on the chest roentgenogram.

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SHAPTINI, E. A.: Byssinosis, a review. *Indust. Med. & Surg.* 30: 95, 1961.

### (9) Bagassosis

Bagasse is the fibrous material remaining after the sugar-containing juice has been expressed from sugar cane. It is used in insulating and acoustic materials as well as in the manufacture of paper, fertilizer, explosives, animal feed, and refractory brick.

Chemically, bagasse consists of approximately 4 percent ash and 2 percent protein with the remaining portion being made up of cellulose and other complex plant carbohydrates and resins.

Bagassosis is a lung disease produced by the inhalation of dust attendant with the opening of bagasse bales which have been stored for several months or years and have subsequently become very dry. Bagasse which is moist from recent grinding, or which has been stored in an unbaled condition, is apparently incapable of producing bagassosis.

Although the specific etiologic mechanism involved in bagassosis is unknown, the following possibilities have been suggested: (1) The disease constitutes an allergic reaction to microorganisms released when the bales are opened, (2) the disease is primarily an infectious process, (3) inhalation of the fibrous bagasse causes irritation of the pulmonary tissues and resultant pathologic changes, (4) irritant products other than the bagasse fibers are released and inhaled with the observed effects, or (5) an interaction of two or more of the above mechanisms.

Clinically, bagassosis presents itself as an acute pneumonitis or bronchitis. In most instances, after exposure to the dust for a few weeks to a few months, symptoms begin to appear over a space of several days. Cough, exertional dyspnea, and low grade fever are usually the initial complaints. Hemoptysis of a mild degree is rather common, but true pulmonary hemorrhage is rare.

As the disease progresses, dyspnea becomes more and more severe, and soon the patient becomes incapacitated. Cyanosis is present in severe cases. Weakness, anorexia, and weight loss are common complaints.

Physical examination may reveal dyspnea, cyanosis, and crepitant rales. Examinations of the sputum generally are negative for pathogenic micro-organisms, including the tubercle bacillus.

Roentgenograms of the chest often disclose the presence of miliary shadows symmetrically distributed throughout both lungs, which appear very similar to the shadows seen in typical miliary tuberculosis. Patchy areas of increased densities suggesting bronchopneumonic infiltration are also seen. Lesions are usually more in evidence in the hilar areas and at the lung bases while the apices are often spared. The cardiac shadow may be enlarged and the pulmonary artery segment may be very prominent.

Pulmonary function studies indicate that there occurs in this disease a disturbance of ventilatory function as shown by a lowered vital capacity and a diminished maximum breathing capacity. These findings are compatible with the presence of airway obstruction such as might be produced by bronchiolitis. In addition, there has been some evidence to indicate the presence of a disturbance of gas exchange at the alveolar capillary level.

The great majority of patients suffering from bagassosis tend to improve spontaneously when they are removed from contact with the offending agent. Symptoms gradually abate over a period of several weeks, and recovery usually takes place in 1 to 6 months. However, some impairment of pulmonary function may be detected for longer periods, and the question of whether there occurs permanent functional lung damage has not yet been answered.

The diagnosis of bagassosis is based on the occupational history and the characteristic, but not pathognomonic, clinical and roentgenographic picture.

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## (10) Farmer's Lung

Farmer's lung is the name given to the disease entity produced by the inhalation of dust arising from moldy silage. Characteristically, symptoms of marked dyspnea, chills, fever, and cough occur several hours following the initial exposure to the dust. The onset of these symptoms may, however, be delayed for several weeks. In addition, auscultation of the chest usually reveals the presence of diffusely scattered, crepitant rales. Wheezes and

ronchi are often present but not invariably so. Dyspnea may be so extreme as to be associated with cyanosis. Weight loss may be pronounced.

Chest roentgenograms taken during this acute phase of the disease generally show changes which parallel the severity of symptoms. Fine to coarse nodular densities may be seen scattered diffusely throughout both lung fields. Conglomeration of these nodules is not an infrequent occurrence. If additional exposure to the dust does not occur, symptoms clear in one or two weeks and recovery is generally complete.

With repeated exposures, dyspnea and cough may become progressively more severe and, if exposures continue, irreversible lung changes may result. These changes, which include interstitial fibrosis and diffuse obstructive emphysema, may produce extreme pulmonary insufficiency with attendant incapacitation. Pulmonary function studies frequently reveal a significant increase in residual volume and functional residual capacity, as well as a moderate decrease in vital capacity and maximum breathing capacity.

The pathogenesis of farmer's lung remains obscure. While fungi seem to play an important etiologic role, the disease is apparently not a true pulmonary mycosis. Available evidence indicates that the pulmonary reaction is due either to one or both of the following: a mechanical irritation produced by the action of dust on lung tissue, or the production of a hypersensitive state by the molds or their disintegration products.

Histologic sections of involved lungs often reveal the presence of acute granulomatous interstitial pneumonitis and, in some cases, interstitial fibrosis, bronchiectasis and emphysema.

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FRANK, R. C.: Farmer's lung, a form of pneumoconiosis due to organic dusts. *Am. J. Roentgenol.* 79: 189, 1958.

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### Some Threshold Limit Values

The threshold limit values carried by the accompanying table were again adopted in 1963 by the American Conference of Governmental Industrial Hygienists. The percent of crystalline silica appearing in the formula, the percent  $\text{SiO}_2$  increased by 5 divided into 250, is derived from an analysis of the air-borne dust.

<i>Substance</i>	<i>Millions of particles per cubic foot of air</i>
<b>SILICA:</b>	
<i>Crystalline:</i>	
Quartz.....	250/(percent $SiO_2 + 5$ ). <i>Do.</i>
Cristobalite.....	20.
<i>Amorphous, including natural diatomaceous earth.</i> .....	
<b>SILICATES:*</b>	
Asbestos.....	5.
Mica.....	20.
Soapstone.....	20.
Talc.....	20.
<b>MISCELLANEOUS*</b> .....	50.

\*Less than 1 percent crystalline silica.



## • section VI

### CHEMICAL HAZARDS

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#### Introduction

Raw materials from oil, gas and salt wells, mines, forests, the sea, air, and farms are converted by the chemical industry into thousands of chemicals such as acids, alkalis, salts, and organic compounds. These chemicals are used by the industry itself to produce cosmetics, detergents and soaps, drugs, dyes, pigments, explosives, fertilizers, petrochemicals, inks, paints, pesticides, plastic materials, synthetic fibers, synthetic rubber, and many other products. Other industries use the chemicals in the production of durable and non-durable goods. *Durable goods* include aircraft and equipment, building materials, electric equipment, hardware, machinery, metal products, motor vehicles and equipment, and other products of metal, glass, paper, and wood. *Nondurable goods* include beverages, food products, leather and leather products, packaging, paper and paper products, petroleum and coal products, rubber products, and textiles.

This section deals with the harmful effects of various substances according to their capacity to produce local and systemic effects; special diagnostic tests that may aid diagnosis, and identification of the agent; the recommended threshold limit of safe exposure (8 hours, daily) when this has been established; potential occupational exposures; and references.

#### *Harmful Effects*

Under the heading, *Harmful Effects*, are given only the chief or dominant effects that characterize the usual response to the toxic agent. Because of the lack of information on the mutagenic effects of chemicals, no consideration is given in specific instances to these effects. Such damage results from the injury of the genetic material of the cells that the chemicals enter. Most of the evidence concerning such effects has been derived from experimental work on microorganisms, plants, and insects. Chemicals in this category are said to be radiomimetic in that they mimic biologic effects usually associated

with ionizing radiation. Among the chemicals used in industry, ethylene oxide and ethylene imine have been referred to as being radiomimetic.

*Local* and *Systemic* under *Harmful Effects* are included in an effort to categorize the effects of the toxic agent. It was arbitrarily decided to limit local effects to the skin, eyes, and mucous membranes of the upper respiratory tract. Systemic effects include the manifestations elicited by the absorption of the toxic agent into the body and its distribution to the internal organs. In addition, systemic effects include the effects of the agent on the tissues of the lower respiratory and gastrointestinal tracts.

*Route of Entry*, when applicable, is intended to supply information on the method by which the toxic agent is most likely to gain entrance into the body when encountered in the industrial environment. Thus, the oral route of entry is listed only for very toxic chemicals such as lead which may be conveyed to the mouth from the hands or cigarettes of the worker, or swallowed from contamination of the nasopharyngeal secretions.

### *Special Diagnostic Tests*

Ordinary tests such as complete blood counts, routine urinalyses, and chest roentgenograms are not included under the heading, *Special Diagnostic Tests*. Similarly, liver and kidney function tests and cutaneous patch tests have not been included, even though they may be of considerable diagnostic importance. It is felt that the reader-physician need not be reminded of the methods for determining abnormalities in the target organs which are mentioned under *Systemic Effects*.

It should be pointed out that many of these special diagnostic tests are difficult to carry out and should be performed only by qualified laboratories. In addition, the fact should be kept in mind that *normal* values may vary, somewhat, even from competent analytical laboratories.

Because of the absence of significant, interpretable information, no reference is made to behavioral patterns of response to toxic agents.

### *Recommended Threshold Limit*

A great deal more is implied in the heading *Recommended Threshold Limit* than the specific assigned Threshold Limit Value appearing in the tentative list published annually by the American Conference of Governmental Industrial Hygienists. In their use, threshold limits are to be considered practical guides in the control of health hazards and should not be regarded as fine lines between safe and dangerous exposure levels. The threshold limit represents a level of exposure at which it is believed, on best available information, almost all workers may be repeatedly exposed day after day throughout their working lifetime without adverse effect on health, or without significant discomfort. In the establishment of threshold limit values, increasing attention is being given to possible long-term genetic

effects, carcinogenic potential, and to the capacity of chemical agents to produce allergic sensitization.

It should be observed that the definition and application of the threshold limit values depend upon the toxicologic action of the substances. Three categories are recognized in the currently (1963) listed substances: (1) Substances whose primary action is rapid, such as irritants; for these substances the limiting value represents a *ceiling*, a limit not to be exceeded for any period however short. Listed substances in this category are preceded by a "C." (2) Substances whose action is prolonged or cumulative; for these substances the threshold value refers to a time-weighted concentration averaged throughout an 8-hour day. Substances in this category comprise the bulk of the list. (3) The third group of substances comprises a small number, mainly carcinogens, for which at present no contact by any route is to be permitted.

It should be observed that when the threshold limit value refers to the time-weighted concentration averaged throughout an 8-hour day, limited fluctuation is permitted *above* the specified value, provided at least an equivalent fluctuation *below* the value obtains. Excessive fluctuations above the value are to be considered indicative of the existence of a hazardous situation, and proper steps should be taken for its control.

It is most important to note that use of the threshold limits to make comparisons between the toxicity or hazard of two substances is improper. The reason for this is that the factors involved in the choice of a limit for one substance may differ from those used in setting the limit for another. For example, the threshold limit for one substance may be based on comfort while the value for another may be based on acute systemic toxicity. Another limit may have incorporated in it a substantial safety factor because of the highly injurious nature of the agent; another limit may have a relatively small safety factor when it is known that a substance can rarely be lethal. Thus, use of the threshold limits to make comparative toxicity or hazard ratings among toxic agents results in erroneous conclusions.

Similarly, it is incorrect to use the threshold limits either unmodified, or modified by the application of a factor, for community air pollution levels. The threshold limits apply to an 8-hour daily workweek of five days and to reasonably healthy adults; they are not applicable to continuous exposures of young and old, the indisposed, and the diseased.

### *Potential Occupational Exposures*

The list of occupations appended to a particular chemical carries occupations in which the workers so engaged are potentially exposed to the toxic agent. Whether the exposure to the toxic agent constitutes a hazard depends upon such factors as concentration of the agent, how the agent is handled and used, duration of exposure, susceptibility of the worker to the agent,

and the health protection practices that might have been adopted by management. Thus, all hazardous situations imply an exposure, but all exposures are not hazardous.

*Symptoms and exposure*—When the problem is encountered of the relationship between the signs and symptoms presented by the worker and the potential toxic exposures in his occupation, the investigator armed with the knowledge of the major ways by which a toxic chemical enters the body, secures factual information on the physical and chemical characteristics of the work environment and the personal hygiene of the worker. At the same time, it is essential to recognize that (1) chemical formulas offer, at most, only rough guides to the prediction of toxic response and (2) the forms of acute and chronic toxicity are so often dissimilar that prediction cannot be made of the nature of chronic toxicity from acute manifestations.

The ordering of the various environmental and clinical observations into a logical causal chain involves all of the difficulties usually inherent in the determination of the cause and effect relationship. The investigation must be carefully and thoughtfully performed. Particular attention must be given all elements believed relevant, the listing of which can flow only from the experience and training of the investigator, a review of the pertinent literature, and, when indicated, consultation with others who may have experienced similar situations. Especially difficult are the separation of the occupational exposures from the nonoccupational ones and any retrospective study required by a long period of latency between exposure to the toxic agent and the recognition of the disease.

### *References to the Literature*

Specific references to the literature will be found appended to most of the chemicals. A list of general references appears at the end of the section.

### *Organization and Selection of Chemicals*

The various chemicals are alphabetically arranged. In a number of instances related substances are presented together, for example, carbonyls, cycloparaffins, and nitroparaffins. Pesticides appear in a separate section. Although plastics and synthetic resins also comprise a single section, most of the components that produce systemic effects are included in the list of chemical hazards.

Most of the known disease-producing chemicals are given. Moreover many materials, even though innocuous, are presented because the question of toxicity may arise during the course of handling or working with these materials. Other compounds, though controversial from the standpoint of toxicity, are included for this reason. Certain chemicals are excluded because of insufficient data.

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The chemicals follow.

### (1) Acetaldehyde

*acetic aldehyde, aldehyde, ethanal, ethyl aldehyde*

#### Harmful Effects

*Local* Liquid and concentrated vapor are irritating to eyes, skin, and mucous membranes of upper respiratory tract. Contact with liquid can sensitize skin.

*Route of Entry* Inhalation of vapor.

*Systemic* Acute effects are secondary to narcotic action and pulmonary irritation, and include drowsiness, unconsciousness, bronchitis, and pulmonary edema. Chronic poisoning has not been reported from inhalation of vapor.

### *Special Diagnostic Test*

Determination of acetaldehyde in blood or urine. *See Von Oettingen, 1958.*

### *Recommended Threshold Limit*

200 parts per million parts of air by volume or 360 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Acetaldehyde workers	2-Methyl-5-ethyl pyridine makers
Acetic acid makers	Mirror silverers
Acetic anhydride makers	Paraldehyde makers
Acrolein makers	Pentaerythritol makers
Aldehyde pumpmen	Perfume makers
Aldol makers	Phenolic resin makers
Butanol makers	Photographic chemical makers
Chloral makers	Resin makers
Disinfectant makers	Rubber makers
Drug makers	Urea resin makers
Dye makers	Varnishers
2-Ethylhexanol makers	Varnish makers
Explosive workers	Vinegar makers
Flavoring makers	Yeast makers
Lacquer workers	

### *References*

HENSON, E. V.: The toxicology of some aliphatic aldehydes. *J. Occup. Med.* 1: 457, 1959.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (2) Acetic Acid

*ethanoic acid, pyroligneous acid*

Vinegar contains 4-6 percent of acetic acid; commercial acetic acid varies in concentration from 6 to 99 percent. Glacial acetic acid contains about 99 percent of the acid.

### *Harmful Effects*

*Local* High concentrations of acetic acid vapor produce conjunctivitis, lacrimation, nasal irritation, and dental erosion. On contact, glacial

acetic acid produces painful cutaneous burns which are slow to heal, corneal burns, conjunctivitis, and iritis. Repeated contact with dilute solutions can produce a hyperkeratotic and fissured dermatitis from primary irritation.

*Route of Entry* Inhalation of vapor.

*Systemic* Irritant effect of high vapor concentrations, if unheeded, can produce bronchitis and pulmonary edema.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

10 parts per million parts of air by volume or 25 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Acetamide makers	Food preservers
Acetanilide makers	Insecticide makers
Acetate ester makers	Ketene makers
Acetate fiber makers	Laundry workers
Acetic acid workers	Methyl ethyl ketone makers
Acetic anhydride makers	Paris green makers
Acetone makers	Photographic chemical makers
Acetyl chloride makers	Plastic makers
Aspirin makers	Rubber makers
Cellulose acetate makers	Stain removers
Drug makers	Textile printers
Dye makers	Tint rinse makers
Ester makers	Vinegar makers
Ethyl alcohol makers	White lead makers

### *References*

HENSON, E. V.: Toxicology of the fatty acids. *J. Occup. Med.* 1: 339, 1959.

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### (3) Acetic Anhydride

*acetic oxide, acetyl oxide, ethanoic anhydride*

### *Harmful Effects*

*Local* Exposure to liquid or concentrated vapor can produce conjunctivitis, photophobia, lacrimation, irritation of nose and throat, and contact dermatitis due to primary irritation.

*Route of Entry* Inhalation of vapor.

*Systemic* Pulmonary irritation can occur but is usually avoided by heeding early warning symptoms resulting from irritation of upper respiratory tract. No chronic systemic effects have been reported.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

5 parts per million parts of air by volume or 20 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Acetanilide makers	Dye makers
Acetate fiber makers	Explosive makers
Acetic acid makers	Flavoring makers
Acetic anhydride workers	Peracetic acid makers
Acetyl chloride makers	Perfume makers
Aspirin makers	Photographic film makers
Cellulose acetate fiber makers	Plastic makers
Drug makers	Textile makers

### (4) Acetone. *See Ketones*

### (5) Acetonitrile

*methyl cyanide, cyanomethane, ethanenitrile*

### *Harmful Effects*

*Local* Contact dermatitis due to primary irritation of either liquid or concentrated vapor.

*Route of Entry* Inhalation of vapor.

*Systemic* Hydrolyzes to cyanide which is detoxified to thiocyanate. Late symptoms may be due to thiocyanate toxicity. Inhalation of high concentrations can produce headache, weakness, shortness of breath, nausea, diarrhea, chest and abdominal pain, gray color, bleeding from mucous membranes, convulsions, coma, and death. Liver and kidney damage may also occur.

### *Special Diagnostic Test*

Determination of blood cyanide, serum and urinary thiocyanate. *See Amdur, 1959.*

### *Recommended Threshold Limit*

40 parts per million parts of air by volume or 70 milligrams per cubic meter of air.

## Potential Occupational Exposures

Acetonitrile workers	Perfume makers
Animal oil processors	Petroleum hydrocarbon purifiers
Drug makers	Tank coaters
Fiber makers	Thiamine makers
Organic chemical synthesizers	Vegetable oil processors

## References

AMDUR, M. L.: Accidental group exposure to acetonitrile; a clinical study. *J. Occup. Med.* 1: 627, 1959.

RIEDERS, F. AND BRIEGER, H.; LEWIS, C. E., AND AMDUR, M. L.: What is the mechanism of toxic action of organic cyanide? *J. Occup. Med.* 3: 482, 1961. Three answers to the question.

## (6) Acetylene

*ethine, ethyne, narcylene*

## Harmful Effects

*Local* None.

*Route of Entry* Inhalation of gas.

*Systemic* In low concentrations, acetylene acts as narcotic. In high concentrations, it decreases available oxygen, thus causing anoxia. Impurities in commercial acetylene, such as arsine, hydrogen sulfide, phosphine, carbon disulfide, and carbon monoxide, may also produce symptoms.

Initial symptoms are rapid respiration and air hunger. Mental alertness and muscular coordination are impaired. Other manifestations include cyanosis, weak and irregular pulse, nausea, vomiting, prostration, impairment of judgment and sensation, loss of consciousness, convulsions, and death.

## Special Diagnostic Test

None.

## Recommended Threshold Limit

Not established.

## Potential Occupational Exposures

Acetaldehyde makers	Carbon black makers
Acetic acid makers	Ceramic makers
Acetone makers	Chloro- derivative makers
Acetylene black makers	Copper purifiers
Acetylene workers	Descalers
Acrylonitrile makers	Drug makers
Alcohol makers	Dye makers
Braziers	Foundry workers
Butadiene makers	Gougers

Hardeners	Oxyacetylene cutters
Heat treaters	Oxyacetylene solderers
Lead burners	Oxyacetylene welders
Metalizers	Rubber makers
Metal refiners	Scarfers
Motor boat fuel makers	Tetrachloroethane makers
Organic chemical synthesizers	Vinyl derivative makers

### Reference

JONES, A. T.: Fatal gassing in an acetylene manufacturing plant. *Arch. Environ. Health* 1: 417, 1960.

### (7) Acridine

*dibenzopyridine, 10-azaanthracene*

### Harmful Effects

*Local* Either solid or vapor can produce contact dermatitis from primary irritation. Photosensitization dermatitis, conjunctivitis, corneal damage, and sneezing have been reported.

*Route of Entry* Inhalation of vapor.

*Systemic* No serious industrial poisonings have been reported. This is probably due to early warning by intense irritation of nose and throat.

### Special Diagnostic Test

Determination of acridine in blood and urine. See Von Oettingen, 1958.

### Recommended Threshold Limit

Not established.

### Potential Occupational Exposures

Acridine makers	Organic chemical synthesizers
Acridine workers	Pipeline workers
Acriflavine makers	Pitch workers
Asphalt workers	Proflavine makers
Coal tar workers	Quinacrine makers
Coke makers	Railroad track workers
Disinfectant makers	Rim steel makers
Drug makers	Road builders
Dye makers	Roofers
Highway maintenance workers	Stack cleaners
Laboratory workers, chemical	Street repairers
Lacrimator makers	Wood preservers
Methionine makers	

## References

BALDI, G.: Occupational pathology from acridine. *Med. Lavoro* 44: 240, 1953.  
 VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

### (8) Acrolein

*acraldehyde, acrylic aldehyde, allyl aldehyde, propenal*

## Harmful Effects

*Local* Either liquid or concentrated vapor produces intense irritation of eyes, nose, throat, and skin.

*Route of Entry* Inhalation of vapor.

*Systemic* Pulmonary edema and narcosis are possible but seldom occur because of warning properties of vapor.

## Special Diagnostic Test

None.

## Recommended Threshold Limit

0.1 part per million parts of air by volume or 0.25 milligram per cubic meter of air.

## Potential Occupational Exposures

Acrolein workers	Lacrimator makers
Alcohol denaturant workers	Linoleum makers
Allyl alcohol makers	Linseed oil workers
Bookbinders	Methionine makers
Coffee roasters	Organic chemical synthesizers
Cooks	Perfume makers
Core makers	Renderers
Diesel engine workers	Resin makers
Drug makers	Rubber makers
Drying oil workers	Soap makers
Fat processors	Textile resin makers
Foundry workers	Tinsmiths
Galvanizers	Tung oil workers
Glycerine workers	Varnish makers
Heat treaters	

## Reference

HENSON, E. V.: The toxicology of some aliphatic aldehydes. *J. Occup. Med.* 1: 457, 1959.

## (9) Acrylonitrile

*vinyl cyanide, cyanoethylene, propene nitrile*

### *Harmful Effects*

*Local* Liquid and high concentrations of vapor are irritating to eyes and nose. Contact with the liquid is irritating to skin and results in blister formation.

*Routes of Entry* Inhalation of vapor and percutaneous absorption of liquid. May be absorbed from contaminated rubber.

*Systemic* In addition to a toxic action from the whole molecule, acrylonitrile may also exert a toxic action by partial in vivo conversion to cyanide. Toxic effects include headache, nausea, weakness, diarrhea, anemia, and jaundice.

### *Special Diagnostic Tests*

Examination of serum and urine for thiocyanate. Spectrographic determination of acrylonitrile in blood. See Lawton et al., 1943; Wilson and McCormick, 1949; Briege et al., 1952, and Elkins, 1959.

### *Recommended Threshold Limit*

20 parts per million parts of air by volume or 45 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Acrylic resin makers	Paper makers
Acrylonitrile workers	Plasticizer makers
Fumigant workers	Plexiglass makers
Grain fumigators	Polymethacrylate resin makers
Leather finish makers	Rubber makers
Lubricating oil additive makers	Safety glass makers
Lucite makers	Synthetic fiber makers
Organic chemical synthesizers	Textile finish makers

### *References*

BRIEGER, H.; RIEDERS, F., AND HODES, W. A.: Acrylonitrile; spectrophotometric determination, acute toxicity and mechanism of action. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 128, 1952.

ELKINS, H. B.: *The Chemistry of Industrial Toxicology*. 2nd ed. John Wiley & Sons, New York, 1959.

LAWTON, A. H.; SWEENEY, T. R., AND DUDLEY, H. C.: Toxicology of acrylonitrile (vinyl cyanide). 3, Determination of thiocyanates in blood and urine. *J. Indust. Hyg. & Toxicol.* 25: 13, 1943.

WILSON, R. H. AND MCCORMICK, W. E.: Acrylonitrile, its physiology and toxicology. *Indust. Med.* 18: 243, 1949.

## (10) Allyl Alcohol

*vinyl carbinol, propenyl alcohol**Harmful Effects*

*Local* Liquid and vapor are highly irritating to eyes and upper respiratory tract. Effects include lacrimation, photophobia, retrobulbar pain, blurring of vision, corneal ulceration, coryza, and headache. Eye irritation is usually delayed in onset and may be prolonged. Skin irritation and burns have occurred from contact with liquid and may also be prolonged.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Local muscle spasms occur at sites of percutaneous absorption. Pulmonary edema, liver and kidney damage, diarrhea, delirium, convulsions, and death have been observed in laboratory animals but have not been reported in man. Vapor has strong warning properties.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

2 parts per million parts of air by volume or 5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Acrolein makers	Herbicide makers
Allyl alcohol workers	Organic chemical synthesizers
Drug makers	Plasticizer makers
Glycerine makers	Resin makers

*Reference*

DUNLAP, M. K.; KODAMA, J. K.; WELLINGTON, J. S.; ANDERSON, H. H., AND HINE, C. H.: The toxicity of allyl alcohol. I, Acute and chronic toxicity. *A.M.A. Arch. Indust. Health* 18: 303, 1958.

## (11) Aluminum and Compounds

*Harmful Effects*

*Local* Aluminum salts are astringent. Contact may harden and tan skin resulting in fissuring. Aluminum chloride may act as sensitizer and produce contact dermatitis.

*Route of Entry* Inhalation of dust or fume.

*Systemic* Pathogenicity of inhaled aluminum dust or fume is controversial. It is likely that effect on lungs is intimately associated with particle size and purity of material involved. See Shaver's Disease, Pneumoconioses section.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Alumina (aluminum oxide) abrasive makers	Glass makers
Aluminum alloy grinders	Ink makers
Aluminum extractors	Laboratory workers, chemical
Aluminum workers	Lithographers
Ammunition makers	Lubricant makers
Ceramic makers	Paint makers
Cosmetic workers	Paper makers
Dye makers	Petroleum refinery workers
Electronic workers	Plastic makers
Fireworks makers	Pottery makers
Foundry workers	Rubber makers
Gem makers	Tannery workers
	Textile workers

*References*

DWORSKI, M.: Prophylaxis and treatment of experimental silicosis by means of aluminum. *A.M.A. Arch. Indust. Health* 12: 229, 1955.

MCLAUGHLIN, A. I. G.; KAZANTZIS, G.; KING, E.; TEARE, D.; PORTER, R. J., AND OWEN, R.: Pulmonary fibrosis and encephalopathy associated with the inhalation of aluminum dust. *Brit. J. Indust. Med.* 19: 253, 1962.

MITCHELL, J.; MANNING, G. B.; MOLYNEUX, M., AND LANE, R. E.: Pulmonary fibrosis in workers exposed to finely powdered aluminum. *Brit. J. Indust. Med.* 18: 10, 1961.

RIDDELL, A. R.: Pulmonary changes encountered in employees engaged in the manufacture of alumina abrasives. Pathologic aspects. *Occup. Med.* 5: 710, 1948.

SHAVER, C. C.: Pulmonary changes encountered in employees engaged in the manufacture of alumina abrasives. Clinical and roentgenologic aspects. *Occup. Med.* 5: 718, 1948.

VORWALD, A. J. (EDITOR): *Pneumoconiosis; Beryllium, Bauxite Fumes, Compensation.* Sixth Saranac Laboratory Symposium, 1947. Paul B. Hoeber, New York, 1950.

## (12) Ammonia

*Harmful Effects*

*Local* Contact with anhydrous liquid ammonia or with aqueous solutions is intensely irritating to mucous membranes, eyes, and skin. Eye symptoms range from lacrimation, blepharospasm, and palpebral edema to corneal ulceration and blindness. There may be corrosive burns of skin or blister formation. Ammonia gas is also irritating to eyes and moist skin.

*Route of Entry* Inhalation of gas.

*Systemic* Mild to moderate exposure to gas can produce headache, salivation, burning of throat, anosmia, perspiration, nausea, vomiting, and substernal pain. Irritation of ammonia gas in eyes and nose is sufficiently intense to compel workers to flee. If escape is not possible, there is irritation of lower respiratory tract with production of cough, glottal edema, pulmonary edema, or respiratory arrest. Bronchitis or pneumonia may follow a severe exposure if patient survives. Urticaria is a rare allergic manifestation from inhalation of gas.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

50 parts per million parts of air by volume or 35 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Acetylene workers	Gas purifiers
Aluminum workers	Gas workers, illuminating
Amine makers	Glass cleaners
Ammonia workers	Glue makers
Ammonium salt makers	Ice cream makers
Aniline makers	Ice makers
Annealers	Ink makers
Boneblack makers	Laboratory workers, chemical
Braziers	Lacquer makers
Bronzers	Latex workers
Calcium carbide makers	Manure handlers
Case hardeners	Metal extractors
Coal tar workers	Metal powder processors
Coke makers	Mirror silverers
Color makers	Nitric acid makers
Corn growers	Organic chemical synthesizers
Cyanide makers	Paper makers
Decorators	Perfume makers
Diazotypy machine operators	Pesticide makers
Drug makers	Petroleum refinery workers
Dye intermediate makers	Photoengravers
Dye makers	Photographic film makers
Electroplaters	Plastic cement mixers
Electrotypes	Pulp makers
Explosive makers	Rayon makers
Farmers	Refrigeration workers
Fertilizer workers	Resin makers
Galvanizers	Rocket fuel makers

Rubber cement mixers	Sugar refiners
Rubber workers	Sulfuric acid workers
Salt extractors, coke oven byproduct	Synthetic fiber makers
uct	Tanners
Sewer workers	Tannery workers
Shellac makers	Urea makers
Shoe finishers	Varnish makers
Soda ash makers	Vulcanizers
Solvay process workers	Water base paint workers
Stablemen	Water treaters
Steel makers	Wool scourers

### (13) Amyl Acetate

*isoamyl acetate, pear oil, banana oil, amyl acetic ester*

#### *Harmful Effects*

*Local* Vapor is irritating to eyes and respiratory tract, and has produced laryngitis and glottal edema. Prolonged contact with liquid produced dry, scaly, and fissured dermatitis.

*Route of Entry* Inhalation of vapor.

*Systemic* Vapor has a narcotic action, and prolonged inhalation can produce fatigue, headache, vertigo, tinnitus, mental confusion, and somnolence. Overexposure is usually prevented by irritant warning property.

#### *Special Diagnostic Test*

None.

#### *Recommended Threshold Limit*

100 parts per million parts of air by volume or 525 milligrams per cubic meter of air.

#### *Potential Occupational Exposures*

Airplane dope makers	Dry cleaners
Amyl acetate workers	Dyers
Art glass workers	Enamelers
Bath sponge makers	Enamel makers
Battery makers, storage	Explosive workers
Bookbinders	Fruit essence makers
Bronzers	Furniture polishers
Bronzing liquid makers	Gilders
Camphor workers	Hefner lamp users
Cutlery makers	Incandescent lamp makers

Incandescent lamp wirers	Polish makers
Jewelers	Rayon makers
Lacquer removers	Rubber buffers
Lacquer workers	Rubber cement workers
Leather makers	Shellackers
Leather mottlers	Shellac makers
Linoleum makers	Shoe factory workers
Motion picture film workers	Shoe finishers
Nitrocellulose workers	Shoe heel coverers, wood
Painters	Shoe polish makers
Paint makers	Silk makers
Paint removers	Smokeless powder makers
Paper makers, coated	Soap makers
Patent leather makers	Stain removers
Pearl makers	Straw hat makers
Penicillin makers	Tannery workers
Perfume makers	Textile finishers
Photoengravers	Textile printers
Photographic film makers	Toy makers
Plastic cement workers	Varnishers
Plastic makers	Varnish makers
Plastic wood workers	Waterproofing makers

### Reference

NELSON, K. W.; EGE, J. F., JR.; ROSS, M.; WOODMAN, L. E., AND SILVERMAN, L.: Sensory response to certain industrial solvent vapors. *J. Indust. Hyg. & Toxicol.* 25: 282, 1943.

### (14) Amyl Alcohol

*fusel oil, grain oil, potato spirit, potato oil*

Depending on source and method of manufacture, one or more isomeric primary, secondary, or tertiary alcohols may be present. When amyl alcohol is prepared by distillation of fusel oil, the chief constituent is isoamyl alcohol.

### Harmful Effects

*Local* Liquid and vapor are irritating to eyes, mucous membranes, and skin.

*Route of Entry* Inhalation of vapor.

*Systemic* Early effects are irritation of nose and throat, followed by nausea, vomiting, facial flushing, headache, double vision, dizziness, and muscular weakness. Prolonged exposures to high concentrations can cause delirium, loss of consciousness, and death.

### *Special Diagnostic Test*

Determination of amyl alcohol content of blood. *See Von Oettingen, 1958.*

### *Recommended Threshold Limit*

(Isoamyl alcohol) 100 parts per million parts of air by volume or 360 milligrams per cubic meter of air

### *Potential Occupational Exposures*

Alcohol distillery workers	Nitrocellulose workers
Amyl acetate makers	Oil processors
Amyl alcohol workers	Ore upgraders
Amyl nitrite makers	Painters
Antifreeze makers	Paint makers
Drug makers	Perfume makers
Explosive makers	Photographic chemical makers
Fat processors	Plastic makers
Flotation workers	Rubber makers
Fruit essence makers	Shoe finishers
Laboratory workers, chemical	Smokeless powder makers
Lacquerers	Varnishers
Lacquer makers	Varnish makers
Mordanters	Wax processors

### *Reference*

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

### (15) Aniline and Other Amino Compounds of Benzene and Its Homologues

*aminobenzene, phenylamine, aniline oil, aminophen, arylamine*

### *Harmful Effects*

*Local* Liquid may occasionally cause allergic contact dermatitis.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Aniline converts hemoglobin to methemoglobin, which causes anoxia and depression of central nervous system. Aniline may also have a direct toxic action causing hypotension and cardiac arrhythmias. Cyanosis is an early sign and is first noticed on lips, fingertips, and ears. Later, there may be headache, nausea, weakness, generalized aching, tachycardia, visual disturbances, mental confusion, and coma. Prolonged or repeated attacks may lead to anemia. Death from a single exposure is due to respiratory paralysis from central nervous system depression. Jaundice, enlargement of liver and spleen, and urinary bladder irritation

have also been reported following severe poisonings. Aniline is not a bladder carcinogen, but several derivatives such as beta-naphthylamine, benzidine and para-aminobiphenyl produce potentially malignant papillomas of the urinary bladder after years of exposure.

### *Special Diagnostic Tests*

Examination of blood for methemoglobin. Examination of erythrocytes for Heinz bodies. Determination of para-aminophenol and diazotizable metabolites in urine. See Hill, 1953; Von Oettingen, 1958, and Elkins, 1959.

### *Recommended Threshold Limit*

(Aniline) 5 parts per million parts of air by volume or 19 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Acetanilide workers	Painters
Acetic anhydride makers	Paint makers
Aniline workers	Paint remover makers
Antifouling paint makers	Pencil makers, colored
Blueprint paper makers	Perfume makers
Bromine makers	Petroleum refinery workers
Camphor makers	Photographic chemical makers
Coal tar workers	Plastic workers
Color makers	Polish makers
Compositors	Printers
Disinfectant makers	Quinone makers
Drug makers	Rocket fuel makers
Dye makers	Rubber chemical makers
Dyers	Rubber mixers
Explosive makers	Rubber reclaimers
Feather workers	Rubber workers
Gasoline blenders	Rubber workers, pressroom
Hydroquinone makers	Tannery workers
Ink makers	Tetryl makers
Leather makers	Textile printers
Lithographers	Varnishers
Millinery workers	Varnish makers
beta-Naphthylamine workers	Vulcanizers
Nitraniline workers	

### *References*

ELKINS, H. B.: *The Chemistry of Industrial Toxicology*. 2nd ed. John Wiley & Sons, New York, 1959.

HALSTED, H. C.: Industrial methemoglobinemia. *J. Occup. Med.* 2: 591, 1960.  
 HILL, D. L.: Excretion of diazotizable metabolites in man after aniline exposure. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 347, 1953.  
 MUNN, A.: An unusual source of aniline poisoning. *Trans. Assoc. Indust. Med. Officers* 7: 78, 1957. Refers to marking ink used on wearing apparel.  
 VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (16) Antimony and Compounds

### *Harmful Effects*

*Local* Antimony and certain of its salts, notably antimony fluoride, antimony trichloride, antimony tartrate, and antimony pentasulfide are irritant to skin and may produce contact dermatitis. Antimony trichloride has been reported to cause irritation and excoriation of mucous membranes of mouth and pharynx as well as swelling and vesiculation of lips and perforation of nasal septum.

*Routes of Entry* Ingestion or inhalation of dust or fume.

*Systemic* Trivalent antimony compounds are many times more toxic than pentavalent derivatives. Ingestion may produce gastrointestinal irritation with nausea, vomiting, and diarrhea. In acute severe poisonings due either to ingestion or inhalation of excessive amounts of antimony, there may be death from circulatory or respiratory failure or, as a late complication, toxic hepatitis proceeding to acute yellow atrophy. Inhalation of antimony dust may cause acute pneumonitis.

Chronic antimony poisoning is similar to chronic arsenic poisoning. There may be lassitude, irritability, stomatitis, nausea, constipation, myalgia, arthralgia, and leukopenia. There is some evidence that the heart may be injured in course of chronic antimony intoxication.

### *Special Diagnostic Tests*

Examination of blood and urine for excessive amounts of antimony. See Elkins, 1959.

### *Recommended Threshold Limit*

(Antimony) 0.5 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Antimony ore smelters	Dye makers
Antimony workers	Fireworks makers
Babbitt metal workers	Flameproofers
Battery workers, storage	Glass makers
Britannia metal workers	Gold refiners
Bronzers	Lake color makers
Cable splicers	Lead burners
Ceramic makers	Lead hardeners

Leather mordanters	Plaster cast bronzers
Match workers	Rubber makers
Metal bronzers	Solder makers
Miners	Textile dyers
Organic chemical synthesizers	Textile flameproofers
Paint makers	Textile printers
Pewter workers	Type metal workers
Pigment makers	Typesetters

## References

ELKINS, H. B.: *The Chemistry of Industrial Toxicology*. 2nd ed. John Wiley & Sons, New York, 1959.

RENES, L. E.: Antimony poisoning in industry. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 7: 99, 1953.

## (17) Arsenic

Arsenic trioxide, the principal form in which the element is used, is frequently designated as arsenic or white arsenic. The element is considered nonpoisonous.

### Harmful Effects

*Local* Contact with arsenic may produce facial and flexural eczematous dermatitis, ulcerations of the skin, conjunctivitis, rhinitis, nasal perforation, folliculitis, and pustules. Most of these effects are due to primary irritation, but some cases of contact dermatitis are due to allergic hypersensitivity. Prolonged absorption may result in generalized "rain drop" hyperpigmentation, premalignant keratoses on palms and soles, hair loss, and nail dystrophy.

*Routes of Entry* Ingestion or inhalation of dust or fume.

*Systemic* Acute systemic poisoning from ingestion produces a violent gastroenteritis, which may be followed by nephritis, hepatitis, or neuritis, but this type of poisoning is rare in industry. A massive inhalation exposure can produce bronchitis, but acute systemic intoxication is unlikely by this route. When arsenical intoxication occurs in industry, it is usually chronic in form. High exposures are frequently tolerated without symptoms of systemic poisoning.

Chronic exposure is characterized by insidious onset of malaise, abdominal complaints, pruritis, weakness, anorexia, and weight loss. There may be gingivitis and stomatitis with *garlic breath*. However, the garlic breath may be due to contamination with tellurium. Peripheral nerve degeneration resulting in progressive sensory alterations and motor disturbances is common. Kidney and liver damage may also occur. Prolonged inhalation of dust may result in laryngitis and bronchitis. Arsenic

has been suspected, but not proved, as a cancer producing agent in the liver and lungs.

### Special Diagnostic Tests

Analysis of urine, hair, or nails for abnormal amounts of arsenic trioxide. The presence of arsenic in urine in amounts greater than 0.2 mg. per liter, is strongly suggestive of excessive absorption. See Elkins, 1959, and Vallee et al., 1960.

### Recommended Threshold Limit

0.5 milligram per cubic meter of air.

### Potential Occupational Exposures

Alloy makers	Insecticide makers
Aniline color makers	Lead shot makers
Arsenic workers	Lead smelters
Babbitt metal workers	Leather workers
Boiler operators	Painters
Brass makers	Paint makers
Bronze makers	Petroleum refinery workers
Bronzers	Pigment makers
Cattle dip workers	Printing ink workers
Ceramic enamel makers	Rodenticide makers
Ceramic makers	Semiconductor compound makers
Copper smelters	Sheep dip workers
Drug makers	Silver refiners
Dye makers	Taxidermists
Enamelers	Textile printers
Farmers	Tree sprayers
Fireworks makers	Type metal workers
Glass makers	Water weed controllers
Gold refiners	Weed sprayers
Hair remover makers	Wood preservative makers
Herbicide makers	Wood preservers
Hide preservers	

### References

DINMAN, B. D.: Arsenic; chronic human intoxication. *J. Occup. Med.* 2: 137, 1960.  
 ELKINS, H. B.: *The Chemistry of Industrial Toxicology*. 2nd ed. John Wiley and Sons, New York, 1959.  
 HOLMQVIST, I.: Occupational arsenical dermatitis; a study among employees at a copper-ore smelting works including investigations of skin reactions to contact with arsenic compounds. *Acta dermat.-venereol. Supp.* 26, 1951.  
 PINTO, S. S. AND MCGILL, C. M.: Arsenic trioxide exposure in industry. *Indust. Med. & Surg.* 22: 281, 1953.  
 VALLEE, B. L.; ULMER, D. D., AND WACKER, W. E. C.: Arsenic toxicology and biochemistry. *A.M.A. Arch. Indust. Health* 21: 132, 1960.

## (18) Arsine

*hydrogen arsenide, arsenic trihydride, arseniuretted hydrogen*

Arsine may be produced wherever nascent hydrogen comes in contact with arsenic. The hydrogen is usually produced by the action of acid upon a metal, the arsenic being present as an impurity in the metal or in the acid.

### *Harmful Effects*

*Local* Bronze discoloration of skin.

*Route of Entry* Inhalation of gas.

*Systemic* Hemolysis of red blood corpuscles with resulting anemia and jaundice. Peripheral neuritis, visual disturbances, and delirium. Chronic intoxication may result in nephritis, myocarditis, and hepatitis. Garlic-like odor may be noted on breath.

### *Special Diagnostic Tests*

Analysis of urine and blood for arsenic. In cases of chronic exposure, analysis of body hair for arsenic. See Elkins, 1959.

### *Recommended Threshold Limit*

0.05 part per million parts of air by volume or 0.2 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Acetylene workers	Lead burners
Acid dippers	Lime burners
Aniline workers	Metal cleaners
Arsine workers	Metal refiners
Bleaching powder makers	Nitrocellulose makers
Bronzers	Ore smelter workers
Cadmium workers	Organic chemical synthesizers
Dimethyl sulfate makers	Paper makers
Dye makers	Petroleum refinery workers
Electrolytic copper makers	Plastic workers
Electroplaters	Plumbers
Etchers	Rayon makers
Ferrosilicon workers	Soda makers
Fertilizer makers	Solderers
Galvanizers	Submarine workers
Gold extractors	Sulfuric acid workers
Hydrochloric acid workers	Tinners
Illuminating gas workers	Zinc chloride makers
Jewelers	

## References

BULMER, F. M. R.; ROTHWELL, H. E.; POLACK, S. S., AND STEWART, D. W.: Chronic arsine poisoning among workers employed in the cyanide extraction of gold; a report of fourteen cases. *J. Indust. Hyg. & Toxicol.* 22: 111, 1940.

ELKINS, H. B.: *The Chemistry of Industrial Toxicology*. 2nd ed. John Wiley & Sons, New York, 1959.

JOSEPHSON, C. J.; PINTO, S. S., AND PETRONELLA, S. J.: Arsine; electrocardiographic changes produced in acute human poisoning. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 43, 1951.

## (19) Barium and Compounds

### Harmful Effects

*Local* The soluble barium salts are irritating to skin and mucous membranes and may produce dermatitis, conjunctivitis, and marked bronchial irritation. Barium sulfide is known for its depilatory and bleaching action.

*Route of Entry* Ingestion or inhalation of dust or fume.

*Systemic* The soluble barium salts are highly toxic. Barium stimulates smooth, striated, and cardiac muscle and may produce violent peristalsis, arterial hypertension, muscle twitching, and cardiac dysfunction.

Barium sulfate is relatively insoluble and therefore innocuous when ingested; however, prolonged inhalation has been reported to cause a benign form of pneumoconiosis known as *baritosis*.

### Special Diagnostic Test

Analysis of urine for barium. See Stewart and Stolman, 1961.

### Recommended Threshold Limit

(Soluble compounds) 0.5 milligram per cubic meter of air.

### Potential Occupational Exposures

Animal oil refiners	Disinfectant makers
Barite millers	Drug makers
Barite miners	Dye makers
Barium workers	Dyers
Bearing packing makers	Electroplaters
Black ash workers	Embalming fluid workers
Boiler operators	Enamel makers
Brick makers	Explosive makers
Ceramic enamel makers	Fat processors
Ceramic makers	Fireworks makers
Core makers	Frit makers
Crystal makers (spectroscope, storage devices, digital calculators)	Fungicide makers
	Glass makers
	Glaziers

Grease additive makers	Plastic makers
Hair removers	Rodenticide makers
Ink makers	Rubber makers
Insecticide makers	Soap makers
Laboratory workers, chemical	Steel carburizers
Lake color makers	Straw hat bleachers
Linoleum makers	Tannery workers
Lithopone makers	Textile bleachers
Luminous paint workers	Textile mordanters
Match makers	Textile printers
Oil additive makers	Tracer bullet makers
Oilcloth makers	Varnish makers
Oil well drillers	Vegetable oil processors
Organic chemical synthesizers	Vulcanizers
Paint makers	Waterproofers
Paper makers	Water treaters
Photographic chemical makers	Wax processors
Pigment makers	

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## (20) Benzene

*benzol, phenyl hydride, coal naphtha, phene, benzole, cyclohexatriene*

### Harmful Effects

*Local* Exposure to liquid or vapor may produce primary irritation of skin, eyes, and mucous membranes of upper respiratory tract. Skin effects may include erythema, vesiculation, or a dry, scaly dermatitis.

*Routes of Entry* Inhalation of vapor. Percutaneous absorption of liquid leading to systemic toxicity is unlikely to occur.

*Systemic* Acute high exposures are responsible for initial exhilaration followed by signs and symptoms of central nervous system depression, including drowsiness, fatigue, headaches, dizziness, loss of consciousness, convulsions, and death.

Chronic low-level exposures may produce alterations of blood elements most commonly resulting in anemia, leukopenia, and thrombocytopenia. The bone marrow effects may be normal, hyperplastic, or hypoplastic and do not necessarily reflect the state of peripheral blood. Symptoms and signs relative to depression of these blood cellular elements include headache, fatigue, dizziness, loss of appetite, weakness, breathlessness, bleeding

from the nose and other mucous membranes, purpura, easy bruising, and proneness to infection. These effects generally improve after removal of the worker from areas of excessive exposure.

Benzene is a suspected carcinogenic agent. All forms of acute and chronic leukemia have been observed in workers with benzene intoxication.

### *Special Diagnostic Tests*

Analysis of urinary sulfate and calculation of the urinary sulfate ratio (inorganic/total sulfate). Analysis of blood and urine benzene or urine phenol may be helpful in evaluating type and degree of exposure. *See Gerarde, 1960.*

### *Recommended Threshold Limit*

25 parts per million parts of air by volume or 80 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Adhesive makers	Drug makers
Airplane dope makers	Dry cleaners
Alcohol workers	Dye makers
Aniline makers	Explosive makers
Art glass workers	Fumigant makers
Asbestos product impregnators	Fungicide makers
Battery makers, dry	Furniture finishers
Belt scourers	Glue makers
Benzene hexachloride makers	Hairdressers
Benzene workers	Herbicide makers
Bronzers	Histology technicians
Burnishers	Ink makers
Can makers	Insecticide makers
Carbolic acid makers	Lacquer makers
Chlorobenzene makers	Leather makers
Coal tar refiners	Linoleum makers
Coal tar workers	Lithographers
Cobblers	Maleic acid makers
Coke oven workers	Millinery workers
Cyclohexane makers	Nitrobenzene makers
DDT makers	Nitrocellulose workers
Degreasers	Oilcloth makers
Detergent makers	Oil processors
Dichlorobenzene makers	Organic chemical synthesizers
Diphenyl makers	Painters

Paint makers	Rubber gasket makers
Paraffin processors	Rubber makers
Pencil makers	Shellac makers
Perfume makers	Solvent makers
Petrochemical workers	Stainers
Petroleum refinery workers	Stain makers
Photographic chemical makers	Styrene makers
Picric acid makers	Synthetic fiber makers
Pottery decorators	Type cleaners
Printers	Varnish makers
Putty makers	Wax makers
Resin makers	Welders
Rubber cementers	Wire insulators

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### (21) Benzidine

*benzidine base, paradiaminodiphenyl*

### Harmful Effects

*Local* Primary irritant contact dermatitis has been reported; allergic contact dermatitis is rare.

*Routes of Entry* Percutaneous absorption, from dust; inhalation of dust, and ingestion.

*Systemic* Benzidine is a urinary bladder carcinogen. The actual carcinogens are probably metabolites, 4,4-diamino-3-diphenyl hydrogen sulfate or the orthohydroxy benzidine. Urinary manifestations are frequency, dysuria, and hematuria. Benzidine is unimportant as a methemoglobin former.

### Special Diagnostic Test

Analysis of quinonizable substances in urine. See Glassman and Meigs, 1951.

### Recommended Threshold Limit

Because of high incidence of bladder tumors in man, any exposure, including skin, is extremely hazardous.

*Potential Occupational Exposures*

Benzidine workers	Dye makers
Biochemists	Microscopists
Congo red makers	Organic chemical synthesizers
Crime detection laboratory workers (blood stains)	Rubber makers
Dye intermediate makers	Wood chemists

*References*

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MEIGS, J. W.; BROWN, R. M., AND SCiarini, L. J.: A study of exposure to benzidine and substituted benzidines in a chemical plant. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 533, 1951.

(22) *Benzyl Chloride**alpha-chlorotoluene**Harmful Effects*

*Local* Both liquid and concentrated vapor are highly irritating to eyes and mucous membranes. In eye, benzyl chloride is not only a potent lacrimator but also a protein denaturant. On skin, liquid is a vesicant.

*Route of Entry* Inhalation of vapor.

*Systemic* Systemic effects are usually prevented by intolerable irritation of eyes and nose. Continued exposure can produce bronchial irritation, cough, and pulmonary edema.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

1 part per million parts of air by volume or 5 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Benzyl chloride workers	Gasoline additive workers
Drug makers	Germicide makers
Dye intermediate makers	Motor fuel blenders
Dye makers	Penicillin makers

Perfume makers

Rubber makers

Photographic developer makers

Tannin makers

Resin makers

Wetting agent makers

### (23) Beryllium and Compounds

Prior to 1949 the fluorescent lamp industry used beryllium in the inside coating of lamps. With the recognition of the ill effects of the metal and its compounds, the industry on June 30, 1949, by general agreement eliminated beryllium by substituting another material.

#### *Harmful Effects*

*Local* Contact with beryllium salts may produce contact dermatitis of the hypersensitivity or primary irritant type. Contamination of abrasions or superficial lacerations with the more soluble beryllium salts may cause a chronic, indolent ulcer. Intracutaneous implantation of spicules of beryllium metal or certain beryllium salts may result in the formation of a low-grade granulomatous lesion. Irritation of conjunctiva and cornea may follow contact with beryllium salts, as may rhinitis and nasopharyngitis.

*Route of Entry* Inhalation of fume or dust.

*Systemic* Inhalation of beryllium dust or fume may result in the production of systemic disease either of an acute or of a chronic nature, depending upon the extent of exposure and the nature of the beryllium compound involved.

Acute beryllium disease has resulted from exposure to beryllium compounds in industrial plants producing beryllium from the ore, in metallurgic and ceramics laboratories, and in the fluorescent lamp industry. The following beryllium compounds, in addition to the metal, have been shown to cause acute poisoning: beryllium oxide, sulfate, fluoride, hydroxide, and chloride. The cases associated with the preparation of phosphors involved exposure to beryllium oxide and to zinc beryllium silicate.

Chronic beryllium poisoning has been reported as resulting from exposure in plants handling beryllium phosphors, in beryllium copper founding, in ceramics laboratories, in metallurgic shops and in plants producing beryllium compounds from the ore. This disease has also been reported as occurring among individuals exposed to atmospheric pollution in the vicinity of plants processing beryllium and in persons dwelling in the same household as beryllium workers. Inhalation of the dust of beryl, the beryllium ore, has produced to date no known cases of acute or chronic beryllium poisoning.

Granulomatous lesions of the skin, liver, kidneys, spleen, and lymph nodes may be seen in some patients with beryllium disease; however,

the most striking features of both the acute and chronic forms are referable to the lungs.

Although of dissimilar roentgenologic and histopathologic appearance, both the acute and the chronic forms of beryllium poisoning have some similar signs and symptoms. These include a relatively nonproductive cough, progressive dyspnea, anorexia, and loss of weight. The chief differences between the two forms are seen in the suddenness of onset and in the rate of progression. In neither the acute nor the chronic form of beryllium disease has there been reported any evidence to suggest that microorganisms might play a significant role in pathogenesis.

In the acute pulmonary form, the symptoms of pneumonitis may appear within several hours to several weeks following the initial exposure of the patient to beryllium, and the radiographic changes may become noticeable within from one to three weeks after the onset of symptoms. There is usually rapid progression of signs and symptoms including dyspnea, anorexia, and extreme weight loss. There is generally complete recovery within a period of about 6 months. Cases which terminate fatally usually do so as a result of acute cor pulmonale.

The typical pattern shown by the chest roentgenogram in acute beryllium pneumonitis is a bilateral, patchy infiltrate which resembles the pattern seen in pulmonary edema. This infiltrate may be superseded by a coarse, nodular appearance before final clearance or recovery occurs.

The pathologic lesion seen in the lung in acute beryllium disease is a chemical pneumonitis or bronchoalveolitis, the severity of which is usually proportional to the intensity of exposure.

In chronic beryllium disease the symptoms are generally delayed in onset and persistent in character. They are commonly precipitated or exacerbated by stresses such as pregnancy, respiratory infection, and thyrotoxicosis. The pulmonary manifestations may be mimicked by symptoms of other lung diseases, such as the fibrosing interstitial pneumonitis of the Hamman-Rich syndrome and the pulmonary granulomatosis of sarcoidosis. Dyspnea, cough, anorexia, and weight loss are among the most frequent manifestations of chronic beryllium disease. As the disease progresses, signs and symptoms of cor pulmonale may supervene.

The earliest roentgenographic evidence of pulmonary involvement may appear within a few weeks of the first symptoms of the disease. The most significant feature of the roentgenogram is a uniform distribution of fine granulation, with variation from a ground glass appearance through a diffuse reticular pattern to distinct nodulation superimposed on a granular background.

Additional aid in the diagnosis of chronic beryllium poisoning may be gained through the study of pulmonary function, by use of the beryllium

patch test, through determinations of the beryllium content of body fluids, and through histologic and chemical study of the surgical lung biopsy.

It is generally accepted that the basic pulmonary physiopathology in this disease is an alveolar-capillary block. This diffusion defect can usually be demonstrated in patients with chronic beryllium disease and, while it is not pathognomonic, it may often be helpful in ruling out certain other of the pulmonary granulomatoses.

The place of the patch test in the diagnosis of beryllium disease is uncertain. Some investigators have shown excellent correlation between positive skin reactions to beryllium and proved poisoning, while others have not been able to show such correlation and have pointed out certain hazards inherent in the test itself.

The finding of increased amounts of beryllium in the body tissues and fluids does not, by any means, justify in itself a diagnosis of beryllium disease, nor does the absence of increased amounts of beryllium rule out chronic beryllium poisoning.

The more liberal application of the use of the surgical lung biopsy has been of major aid in the diagnosis of beryllium disease. It must be pointed out, however, that in some cases even the most experienced pathologist may find it impossible to distinguish between this condition and sarcoidosis by examination of histologic sections.

There is no available evidence to implicate beryllium disease as predisposing to pulmonary tuberculosis. Moreover, a causal relationship between beryllium disease and lung cancer has not been established.

### *Special Diagnostic Tests*

Analysis of urine and tissue for abnormal amounts of beryllium. *See Cholak, 1959.*

### *Recommended Threshold Limit*

(Beryllium) 0.002 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Beryllium alloy machiners	Electric equipment makers
Beryllium alloy makers	Fluorescent screen makers
Beryllium compound makers	Gas mantle makers
Beryllium copper founders	Missile technicians
Beryllium copper grinders	Neon sign workers
Beryllium copper polishers	Neon tube makers
Beryllium extractors	Nonsparking tool makers
Beryllium metal machiners	Nuclear physicists
Beryllium phosphor makers	Nuclear reactor workers
Beryllium workers	Precision instrument makers
Cathode ray tube makers	Refractory material makers
Ceramic makers	

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## (24) Bismuth and Compounds

### Harmful Effects

*Local* Bismuth subnitrate may cause skin irritation.

*Route of Entry* Ingestion of powder.

*Systemic* Basic salts are insoluble and exhibit low oral toxicity. Formerly used in an injectable form as a treatment for syphilis. Toxic symptoms following injection include loss of appetite, foul breath, gingivitis, stomatitis, weakness, and diarrhea. Toxic hepatitis and nephritis rarely occur. No poisonings related to occupation have been found in the literature.

### Special Diagnostic Tests

Analysis of blood and urine for excessive amounts of bismuth. *See* Von Oettingen, 1958.

### Recommended Threshold Limit

Not established.

## Potential Occupational Exposures

Bismuth workers	Laboratory workers, chemical
Ceramic capacitor makers	Luminous enamel makers
Ceramic colorers	Luminous paint makers
Ceramic enamel makers	Metallic bath workers
Cosmetic makers	Pearl makers
Deodorant makers	Perfume makers
Disinfectant makers	Permanent magnet makers
Drug makers	Pigment makers
Dyers	Semiconductor makers
Face powder makers	Solder makers
Fuse makers	Tin lusterers
Fusible alloy makers	

## References

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 VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (25) Boron Compounds

### Harmful Effects

*Local* Boric acid may produce primary skin irritation and conjunctivitis.  
*Routes of Entry* Percutaneous absorption of liquid; inhalation of gas or vapor.

*Systemic* Boron hydrides (diborane, pentaborane, decaborane) are the most important compounds of this group.

Inhalation of diborane may result in chest tightness, cough, headaches, nausea, chills, dizziness, and drowsiness. These complaints are generally of short duration. Pneumonia may develop following severe exposures.

Pentaborane and decaborane produce predominantly central nervous system symptoms and signs. Hyperexcitability, headaches, muscle twitching, convulsions, dizziness, disorientation, and unconsciousness may occur early or be delayed for 24 hour or more following excessive exposures to these compounds.

Skin and respiratory tract irritation and central nervous system effects have been reported from animal experiments with amine and alkyl boranes. The alkyl boranes seem to be more toxic than the amine compounds and decaborane, but less toxic than pentaborane. The major effect of repeated inhalation of boron trifluoride in laboratory animals was respiratory irritation which resulted in a pneumonitis.

### Special Diagnostic Tests

Analysis of boron in blood, urine and body tissues. See Jacobson, 1958.

*Recommended Threshold Limit*

Diborane, 0.1 part per million parts of air by volume or 0.1 milligram per cubic meter of air.

Pentaborane, 0.005 part per million parts of air by volume or 0.01 milligram per cubic meter of air.

Decaborane, 0.05 part per million parts of air by volume or 0.3 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

Boron trifluoride, 1 part per million parts of air by volume or 3 milligrams per cubic meter of air.

*Potential Occupational Exposures**Diborane*

Diborane workers  
Organic chemical synthesizers  
Rocket fuel handlers  
Rocket fuel makers

*Pentaborane*

Gasoline additive makers  
Pentaborane workers  
Rocket fuel handlers  
Rocket fuel makers

*Decaborane*

Chemical scavenger makers	Resin makers
Chemical stabilizer makers	Rocket fuel handlers
Decaborane workers	Rocket fuel makers
Dyers	Rubber makers
Gasoline additive makers	Rust inhibitor makers
Insecticide makers	Welding flux makers
Organic chemical synthesizers	

*Boron trifluoride*

Boron trifluoride workers	Nuclear instrument makers
Fumigant makers	Organic chemical synthesizers
Fumigators	

*References*

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 TORKELSON, T. R.; SADEK, S. E., AND ROWE, V. K.: The toxicity of boron trifluoride when inhaled by laboratory animals. *Am. Indust. Hyg. Assoc. J.* 22: 263, 1961.

### (26) Brass

#### *Harmful Effects*

*Local* Brass dust and slivers may cause dermatitis by mechanical irritation.

*Route of Entry* Inhalation of fume.

*Systemic* Brass is chiefly an alloy of copper and zinc, usually in the ratio of 2 to 1. Since zinc melts at a lower temperature than copper, the fusing of brass is attended by liberation of considerable quantities of zinc oxide. Inhalation of zinc oxide fumes may result in production of signs and symptoms of metal fume fever; *see Zinc and Compounds*. *Brass founder's ague* is the name often given to metal fume fever occurring in the brass-founding industry.

Since brass may contain significant quantities of lead, brass foundries may release sufficient amounts of lead fumes into working environment to produce lead intoxication in those workers exposed.

#### *Special Diagnostic Test*

None. *See Lead and Compounds*.

#### *Recommended Threshold Limit*

Not established. Zinc oxide, 5 milligrams per cubic meter of air; lead, 0.2 milligram per cubic meter of air.

#### *Potential Occupational Exposures*

Bench molders	Galvanizers
Brass founders	Junk metal refiners
Brass workers	Welders
Braziers	Zinc founders
Bronzers	Zinc smelters
Core makers	

#### *Reference*

HAMILTON, A. AND HARDY, H. L.: *Industrial Toxicology*. Paul B. Hoeber, New York, 1949.

### (27) Bromine and Compounds

Compounds include hydrogen bromide, methyl bromide (bromomethane), and ethyl bromide (bromoethane). Ethylene dibromide is presented separately.

### *Harmful Effects*

*Local* Bromine and most of its compounds are highly irritating to eyes, mucous membranes of nose and throat, and to skin.

*Routes of Entry* Inhalation of vapor and gas. Percutaneous absorption of methyl bromide may occur.

*Systemic* Acute exposure to high concentrations of bromine or hydrogen bromide can produce pulmonary edema, which may be delayed. There may be a bromine odor on breath.

In acute methyl bromide poisoning, the central nervous system, lungs, liver, and kidney are damaged but effects may be delayed hours after exposure. Pulmonary effects include bronchitis and pulmonary edema. Neurologic effects include headache, visual disturbances, speech dysfunction, mental aberrations, tremors, incoordination, convulsions, and coma. Death is not uncommon. In chronic poisoning the damage is usually limited to central nervous system.

Ethyl bromide is less toxic than methyl bromide but produces similar effects. In addition ethyl bromide has a pronounced narcotic effect and can damage heart.

### *Special Diagnostic Tests*

Blood and urinary bromide determinations. See Von Oettingen, 1958, and Rathus and Landy, 1961.

### *Recommended Threshold Limit*

Bromine, 0.1 part per million parts of air by volume or 0.7 milligram per cubic meter of air.

Hydrogen bromide, 3 parts per million parts of air by volume or 10 milligrams per cubic meter of air.

Methyl bromide, 20 parts per million parts of air by volume or 80 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

Ethyl bromide, 200 parts per million parts of air by volume or 890 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

#### *Bromine*

Bromine workers

Drug makers

Dye makers

Ethylene bromide makers

Ethylene dibromide makers

Fire extinguisher fluid makers

Gold extractors

Methyl bromide makers

Photographic chemical makers

Silk bleachers

Water treaters

Wool shrinkproofers

*Hydrogen bromide*

Barbiturate makers  
Bromide makers  
Drug makers  
Hormone makers

Hydrogen bromide workers  
Organic chemical synthesizers  
Petroleum refinery workers

*Methyl bromide*

Color makers  
Drug makers  
Dye makers  
Fire extinguisher workers  
Fumigant makers  
Grain fumigators  
Insecticide makers

Ionization chamber workers  
Organic chemical synthesizers  
Refrigerant makers  
Refrigeration workers  
Soil fumigators  
Vegetable oil extractors  
Wool degreasers

*Ethyl bromide*

Anesthetists  
Drug makers  
Ethyl bromide workers  
Fruit fumigators  
Fumigant makers

Grain fumigators  
Organic chemical synthesizers  
Refrigerant makers  
Refrigeration workers  
Solvent workers

*References*

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RATHUS, E. M. AND LANDY, P. J.: Methyl bromide poisoning. *Brit. J. Indust. Med.* 18: 53, 1961.

VON OETTINGEN, W. F.: The halogenated aliphatic, olefinic, cyclic, aromatic, and aliphatic-aromatic hydrocarbons including the halogenated insecticides, their toxicity and potential dangers. Pub. Health Service Pub. No. 414. U.S. Government Printing Office, Washington, D.C., 1955.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (28) Butadiene

*biethylene, bivinyl, butadiene monomer, divinyl, erythrene, methylallene, pyrrolylene, vinylethylene*

*Harmful Effects*

*Local* Gas is irritating to mucous membranes and eyes, and liquid can produce a primary irritant type of contact dermatitis.

*Route of Entry* Inhalation of gas.

*Systemic* In high concentrations, gas can act as irritant, producing cough, and as narcotic, producing fatigue, drowsiness, headache, vertigo, loss of consciousness, respiratory paralysis, and death. Probably no cumulative effects.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

1,000 parts per million parts of air by volume or 2,200 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Butadiene workers

Rocket fuel makers

Organic chemical synthesizers

Rubber makers

Rocket fuel handlers

*Reference*

CARPENTER, C. P.; SHAFFER, C. B.; WEIL, C. S., AND SMYTH, H. F., JR.: Studies on the inhalation of 1,3-butadiene; with a comparison of its narcotic effect with benzol, toluol, and styrene, and a note on the elimination of styrene by the human. *J. Indust. Hyg. & Toxicol.* 26: 69, 1944.

(29) Butanone. *See Ketones*

(30) n-Butyl Acetate

*butyl ethanoate, acetic acid butyl ester*

*Harmful Effects*

*Local* High vapor concentrations irritate mucous membranes of eyes, nose, and throat. Vacuolization of corneal epithelium has been observed. Repeated contact with liquid can produce dry, scaly, and fissured dermatitis.

*Route of Entry* Inhalation of vapor.

*Systemic* Based on animal studies, vapor has narcotic effect in high concentrations and can cause drowsiness and loss of consciousness.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

200 parts per million parts of air by volume or 950 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Airplane dope makers

Flavoring makers

n-Butyl acetate workers

Lacquerers

Cellulose acetopropionate  
finishers

Lacquer makers

Dope workers

Leather dope workers

Enameled leather makers

Paper makers, coated

Enamel workers

Patent leather makers

Perfume makers

Photographic film makers	Stain makers
Plastic workers	Stain removers
Safety glass makers	Varnish workers
Stainers	Vinyl resin workers

### Reference

VON OETTINGEN, W. F.: The aliphatic acids and their esters: toxicity and potential dangers; the saturated monobasic aliphatic acids and their esters. *A.M.A. Arch. Indust. Health* 21: 28, 1960.

### (31) n-Butyl Alcohol

*1-butanol, butyl hydroxide, propylcarbinol, butyric alcohol, hydroxybutane*

#### Harmful Effects

*Local* Vapor is an irritant to conjunctiva and mucous membranes of upper respiratory tract. A peculiar keratitis characterized by numerous vacuoles has been reported. Liquid is primary skin irritant.

*Route of Entry* Inhalation of vapor.

*Systemic* No cases of systemic toxicity in humans have been reported, either from n-butyl alcohol or its isomers.

#### Special Diagnostic Test

None.

#### Recommended Threshold Limit

100 parts per million parts of air by volume or 300 milligrams per cubic meter of air.

#### Potential Occupational Exposures

Butyl acetate makers	Nitrocellulose makers
n-Butyl alcohol workers	Photographic film makers
Butyric acid makers	Plasticizer makers
Detergent makers	Polyvinyl resin makers
Di-n-butyl phthalate makers	Rubber cement makers
Dye makers	Shellac makers
Hydraulic fluid makers	Stainers
Lacquerers	Stain makers
Lacquer makers	Urea-formaldehyde resin makers
Melamine resin makers	Varnish makers

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HENSON, E. V.: The toxicology of some aliphatic alcohols; part 2. *J. Occup. Med.* 2: 497, 1960.

STERNER, J. H.; CROUCH, H. W.; BROCKMYRE, H. F., AND CUSACK, M.: A ten-year study of butyl alcohol exposure. *Am. Indust. Hyg. Assoc. Quart.* 10: 53, 1949.

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VON OETTINGEN, W. F.: The aliphatic alcohols, their toxicity and potential dangers in relation to their chemical constitution and their fate in metabolism. Pub. Health Bull. No. 281. U.S. Government Printing Office, Washington, D.C., 1943.

(32) n-Butylamine

*1-aminobutane*

*Harmful Effects*

*Local* Liquid is irritating and produces severe contact dermatitis and corneal injury.

*Route of Entry* Inhalation of vapor.

*Systemic* Vapor is irritating to respiratory tract and can produce pulmonary edema. Stimulation of central nervous system, followed by depression, convulsions, and narcosis.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

5 parts per million parts of air by volume or 15 milligrams per cubic meter of air.

*Potential Occupational Exposures*

n-Butylamine workers	Insecticide makers
Butylaminophenol makers	Petroleum dewaxers
Drug makers	Rubber makers
Dye makers	Tanning chemical makers
Emulsifier makers	

(33) Butyl Mercaptan. *See* Mercaptans

(34) Cadmium

*Harmful Effects*

*Local* Irritant to mucous membranes. Produces yellow discoloration of teeth. Certain salts may cause contact dermatitis due to allergic hypersensitization.

*Routes of Entry* Ingestion or inhalation of fume or dust.

*Systemic* Ingestion results in production of signs and symptoms of acute gastroenteritis. Inhalation of cadmium oxide fume may cause respiratory tract irritation with attendant sore, dry throat and a metallic taste followed by cough, chest pain, and dyspnea. Bronchitis, pneumonitis, and pulmonary edema may occur as result of irritative action of fume. Additional complaints of headache, dizziness, loss of appetite and weight loss may be pronounced. Liver, kidneys, and bone marrow may be injured by the metal.

It is probable that cadmium, under certain conditions, can produce chronic intoxication. Reports suggest that at least 2 years of exposure are necessary for this type of poisoning to develop. The most commonly accepted manifestations of prolonged exposure to cadmium are pulmonary emphysema, renal damage, and proteinuria. The last is not necessarily a result of renal damage and often may be demonstrated in exposed workers with apparently healthy kidneys. Other conditions that have been reported following long exposure to cadmium include anosmia, an increased incidence of nephrolithiasis, and occasional evidence of liver damage.

### *Special Diagnostic Test*

Analysis of urine for increased amounts of cadmium. See Elkins, 1959.

### *Recommended Threshold Limit*

(Cadmium oxide fume) 0.1 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Alloy makers	Incandescent lamp makers
Aluminum solder makers	Lithographers
Battery makers, storage	Lithopone makers
Cadmium compound collecting bag cleaners	Metalizers
Cadmium compound collecting bag handlers	Paint makers
Cadmium platers	Paint sprayers
Cadmium smelters	Photoelectric cell makers
Cadmium vapor lamp makers	Pigment makers
Cadmium workers	Small arms ammunition makers
Ceramic makers	Smoke bomb makers
Dental amalgam makers	Solderers
Electric instrument makers	Solder makers
Electroplaters	Textile printers
Engravers	Welders, cadmium alloy
Glass makers	Welders, cadmium plated object
	Zinc refiners

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TAYLOR, C. M.: Cadmium as a health hazard. *Trans. Assoc. Indust. Med. Officers* 7: 122, 1957.

### (35) Calcium Cyanamide

*nitrolim, calcium carbimide, cyanamide*

#### *Harmful Effects*

*Local* Contact with cyanamide lumps or powder can cause dermatitis from primary irritation or allergic hypersensitivity. Dust can cause irritation of conjunctiva and mucous membranes of nose and throat. Perforation of nasal septum can occur.

*Route of Entry* Inhalation of dust.

*Systemic* Transient attacks of vasodepression manifested by flushing of skin of face, neck, and hands, sweating, hypotension, tachycardia, dyspnea, headache, vertigo, vomiting, and tremors. Symptoms are aggravated by ingestion of alcohol.

#### *Special Diagnostic Tests*

Analysis of blood and urine for cyanamide. Blood spectrum may resemble cyanhemoglobin or cyanhematin. See Von Oettingen, 1958, and Buyske and Downing, 1960.

#### *Recommended Threshold Limit*

Not established.

#### *Potential Occupational Exposures*

Ammonia makers	Herbicide workers
Calcium cyanamide workers	Nitrogen compound makers
Cyanamide makers	Organic chemical synthesizers
Farmers	Steel carburizers
Fertilizer workers	Steel casehardeners

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VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

### (36) Calcium Oxide

*lime, burnt lime, quicklime, calx, fluxing lime*

#### *Harmful Effects*

*Local* Calcium oxide is irritating to skin, conjunctiva, cornea, and mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of dust.

*Systemic* A few cases of pneumonia presumed to be due to inhalation of dust have been reported.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

(Tentative) 5 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Brick masons	Insecticide workers
Calcium carbide makers	Metal smelters
Calcium cyanamide makers	Mortar workers
Calcium oxide workers	Paint makers
Candle makers	Paper hangers
Cement workers	Paper makers
Ceramic workers	Petroleum refinery workers
Chloride of lime makers	Plaster makers
Dye makers	Rubber makers
Electroplaters	Soap makers
Farmers	Steel workers
Fertilizer makers	Sugar refiners
Food processors	Tannery workers
Fungicide workers	Water treaters
Glass makers	

## (37) Carbon Dioxide

*carbonic acid gas*

### *Harmful Effects*

*Local* When carbon dioxide combines in high concentrations with water, carbonic acid is formed. This material upon contact may produce slight skin, eye, or mucous membrane irritation.

*Route of Entry* Inhalation of gas.

*Systemic* Inhalation of 3 to 5 percent carbon dioxide will generally produce an increase in respiratory rate. Concentrations of 8 to 15 percent when inhaled may produce headache, dizziness, nausea, vomiting, and unconsciousness. Exposure to higher concentrations may cause immediate collapse, coma, and death.

Because carbon dioxide is heavier than air, it tends to accumulate in high concentrations in lowermost portions of enclosed or walled-in areas such as at bottom of fermenting tanks or shipholds. This is more likely to occur when the gas is being continuously formed, as during fermenting processes. Because of this tendency, carbon dioxide may dilute normal air in these enclosed areas and act as simple asphyxiant.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

5,000 parts per million parts of air by volume or 9,000 milligrams per cubic foot of air.

*Potential Occupational Exposures*

Aerosol packagers	Foundry workers
Alkali salt makers	Furnace workers
Bakers	Glue makers
Baking powder makers	Grain elevator workers
Beverage carbonators	Ice cream makers
Blast furnace workers	Insecticide makers
Boiler room workers	Lime kiln workers
Brass founders	Linseed oil boilers
Brewers	Mineral water bottlers
Brick burners	Miners
Bronze founders	Natural carbon dioxide workers
Caisson workers	Pottery workers
Canners	Refrigerating car workers
Carbonated water makers	Refrigerating plant workers
Carbon dioxide makers	Salicylic acid makers
Carbon dioxide workers	Sewer workers
Carbonic acid makers	Silo cleaners
Cave explorers	Soda makers
Charcoal burners	Starch makers
Cupola men	Submarine crewmen
Dairy farmers	Sugar refiners
Disinfectant makers	Tannery pit men
Divers	Tobacco moisteners, storehouse
Dock workers	Tunnel workers
Drug makers	Urea makers
Dry ice workers	Vatmen
Drying room workers	Vault workers
Dye makers	Vinegar makers
Ensilage diggers	Vulcanizers
Explosive makers	Welders, inert atmosphere
Fertilizer workers	Well cleaners
Fire extinguisher makers	White lead makers
Firemen	Yeast makers

*Reference*

WILLIAMS, H. I.: Carbon dioxide poisoning; report of eight cases with two deaths. *Brit. Med. J.* 2: 1012, 1958.

## (38) Carbon Disulfide

*carbon bisulfide, dithiocarbonic anhydride**Harmful Effects*

*Local* Liquid and concentrated vapor are irritating to eyes, nose, and skin. Carbon disulfide is one of the most severe of organic solvents in its irritating action on skin.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid or vapor.

*Systemic* Carbon disulfide is potent narcotic agent. Signs and symptoms of acute carbon disulfide poisoning stem from its narcotic action.

In chronic carbon disulfide poisoning, the nervous system bears the brunt of damage. There may be neuritis involving peripheral and cranial nerves (optic and retrobulbar neuritis). Transient mental aberrations are common. These may include mania, depression, hallucinations, and other abnormal mental states. Gastric disturbances are common, and symptoms may simulate those complained of by patients with peptic ulcers. Heart, liver, and kidney damage may result from chronic intoxication.

*Special Diagnostic Tests*

Analysis of urine and blood for carbon disulfide. See Von Oettingen, 1958.

*Recommended Threshold Limit*

20 parts per million parts of air by volume or 60 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Acetylene workers	Explosive workers
Ammonium salt makers	Fat processors
Bromine processors	Flotation agent makers
Carbanilide makers	Fumigant workers
Carbon disulfide workers	Glass makers
Carbon tetrachloride makers	Glue workers
Cellophane makers	Iodine processors
Cementers, rubber shoe	Laboratory workers, chemical
Cement mixers, rubber	Lacquer makers
Coal tar distillers	Match makers
Degreasers	Oil processors
Dry cleaners	Optical glass makers
Dyestuff makers	Painters
Electroplaters	Paint makers
Enamelers	Paint remover makers
Enamel makers	Paraffin workers

Pesticide makers	Smokeless powder makers
Phosphorus processors	Soil fumigators
Preservative makers	Sulfur processors
Putty makers	Tallow makers
Rayon makers	Textile makers
Resin makers	Vacuum tube makers
Rocket fuel makers	Varnish makers
Rubber cement makers	Varnish remover makers
Rubber dryers	Veterinarians
Rubber makers	Vulcanizers
Rubber reclaimers	Wax processors
Selenium processors	

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VIGLIANI, E. C.: Clinical observations on chronic carbon disulfide intoxication in Italy. *Indust. Med. & Surg.* 19: 240, 1950.

## (39) Carbon Monoxide

### Harmful Effects

*Local* None.

*Route of Entry* Inhalation of gas.

*Systemic* Combines with hemoglobin to form carboxyhemoglobin which interferes with oxygen carrying capacity of blood, resulting in a state of tissue hypoxia. Except for this, carbon monoxide is essentially a physiologically inert gas. It is probable that exposure to carbon monoxide gas does not produce a truly chronic type of intoxication but may, upon repeated intermittent exposures, produce repeated transient episodes of mild acute poisoning.

### Special Diagnostic Test

Analysis of blood for carboxyhemoglobin. See Von Oettingen, 1958.

### Recommended Threshold Limit

100 parts per million parts of air by volume or 110 miligrams per cubic meter of air.

### Potential Occupational Exposures

Acetic acid makers	Automobile users
Acetylene workers	Blast furnace gas users
Airplane pilots	Blast furnace workers
Ammonia makers	Boiler room workers
Artificial gas workers	Brass founders

Brewers	Methanol makers
Brick burners	Miners
Carbon monoxide workers	Mond process workers
Diesel engine operators	Nickel refiners
Dock workers	Nickel smelters
Firemen	Organic chemical synthesizers
Foundry workers	Oxalic acid makers
Furnace starters	Producer gas workers
Garage mechanics	Steel makers
Gasoline engine testers	Tunnel attendants
Heat treaters	Water gas workers
Lift truck operators	Zinc white makers
Metal oxide reducers	

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ZORN, O. AND KRUGER, P. D.: The problem of chronic carbon monoxide poisoning. *Indust. Med. & Surg.* 29: 580, 1960.

## (40) Carbon Tetrachloride

*tetrachloromethane, perchloromethane*

### Harmful Effects

**Local** Repeated or prolonged contact with liquid can produce a dry, scaly, fissured dermatitis.

**Routes of Entry** Ingestion of liquid; inhalation of vapor. Percutaneous absorption of liquid leading to systemic intoxication is unlikely to occur.

**Systemic** Excessive exposure will result initially in gastrointestinal irritation or central nervous system depression or both. After a few hours to several days following exposure, signs and symptoms of liver and kidney damage may develop. Nausea, vomiting, abdominal pain, diarrhea, enlarged and tender liver, jaundice, and abnormal liver function tests result from toxic hepatitis. Pulmonary and peripheral edema, elevated blood

pressure, diminished urinary volume, abnormal urinalysis, coma, and death may be the consequence of acute renal failure.

Headache, loss of appetite, and lassitude are characteristic of chronic exposure to carbon tetrachloride.

### *Special Diagnostic Test*

Determination of carbon tetrachloride in blood. *See* Von Oettingen, 1958, and Stewart et al., 1960.

### *Recommended Threshold Limit*

10 parts per million parts of air by volume or 65 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Carbon tetrachloride workers	Lacquer removers
Degreasers	Metal cleaners
Dry cleaners	Oil processors
Fat processors	Propellant makers
Fire extinguisher makers	Refrigerant makers
Fire extinguisher testers	Rotenone extractors
Firemen	Rubber makers
Freon makers	Seed oil extractors
Fumigant makers	Semiconductor makers
Fur storage workers	Solvent workers
Grain fumigators	Stainers
Ink makers	Stain makers
Insecticide makers	Type cleaners
Laboratory workers, chemical	Varnish removers
Lacquerers	Wax makers
Lacquer makers	

### *References*

LEWIS, C. E.: The toxicology of carbon tetrachloride. *J. Occup. Med.* 3: 82, 1961.  
 STEWART, R. D.; TORKELSON, T. R.; HAKE, C. L., AND ERLEY, D. S.: Infrared analysis of carbon tetrachloride and ethanol in blood. *J. Lab. & Clin. Med.* 56: 148, 1960.  
 VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

### (41) Carbonyls

*See also* Nickel and Compounds, and Nickel Carbonyl.

### *Harmful Effects*

*Local* Contact dermatitis from nickel carbonyl, possibly allergic, has been reported.

*Route of Entry* Inhalation of vapor.

*Systemic* In acute intoxications, nickel carbonyl (1) exerts a toxic action on central nervous system with early production of frontal headache, dizziness, nausea, and vomiting and (2) irritates the lungs, producing delayed pneumonitis and pulmonary edema. Retrosternal pain and increased respiratory rate are indications of the delayed pulmonary complications. Metallic taste is occasionally noted. Diarrhea and abdominal distension also occur as a delayed effect. Nickel carbonyl degenerates into metallic nickel and carbon monoxide. The deposition of finely divided nickel within the lungs is thought to be responsible for the pneumonitis and pulmonary edema. The formation of carbon monoxide within the lungs is not thought to be important in the pathogenesis of nickel carbonyl toxicity. Since carbon monoxide is given off from nickel carbonyl in the Mond process of nickel refining, carbon monoxide poisoning may also occur. Allergic bronchial asthma and Loeffler's syndrome have been reported from exposure to nickel carbonyl. Chronic intoxication from nickel carbonyl has not been reported. There is a high incidence of carcinoma of the respiratory tract among nickel refiners, but nickel dust is a more likely carcinogen than nickel carbonyl vapor.

Except for nickel carbonyl no cases of human toxicity to the other existing carbonyls have been reported. Chromium, cobalt, radium, iron, molybdenum, osmium, iridium, rhenium, ruthenium, and tungsten carbonyls exist. Iron and cobalt carbonyls have been shown in animals to be pulmonary irritants. Until further toxicologic information on the lesser known carbonyls becomes available, extreme caution should be exercised with their use.

### *Special Diagnostic Tests*

None, except for analysis of blood and urine for nickel in nickel carbonyl intoxications. See Kincaid et al., 1956.

### *Recommended Threshold Limit*

(Nickel carbonyl) 0.001 part per million parts of air by volume or 0.007 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Acetylene welders	Mond process workers
Blast welders	Nickel refiners
Blast furnace workers	Organic chemical synthesizers
Carbonyl workers	Petroleum refinery workers
Metal refiners	

### *References*

KINCAID, J. F.; STRONG, J. S., AND SUNDERMAN, F. W.: Toxicity studies of cobalt carbonyls. *A.M.A. Arch. Indust. Hyg. & Occup. Med.*, 10: 210, 1954.

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SUNDERMAN, F. W. AND SUNDERMAN, F. W., JR.: Loeffler's syndrome associated with nickel sensitivity. *Arch. Int. Med.* 107: 405, 1961.

#### (42) Cellosolve®

*Cellosolve*: ethylene glycol monoethyl ether, 2-ethoxyethanol

*Cellosolve acetate*: ethylene glycol monoethyl ether acetate, 2-ethoxyethyl acetate

*Methyl cellosolve*: ethylene glycol monomethyl ether, 2-methoxyethanol

*Methyl cellosolve acetate*: ethylene glycol monomethyl ether acetate, 2-methoxyethyl acetate

*Butyl cellosolve*: ethylene glycol monobutyl ether, 2-butoxyethanol

#### *Harmful Effects*

*Local* Contact dermatitis from primary irritation. Vapors are mild irritants to conjunctiva and upper respiratory tract.

*Route of Entry* Inhalation of vapor, and percutaneous absorption of liquid.

*Systemic* Both neurologic and hematologic effects may be seen in methyl cellosolve intoxication; the former are more pronounced in acute exposures, and the latter are more pronounced in low-grade chronic exposures. Neurologic effects include headache, drowsiness, fatigue, forgetfulness, personality aberrations, dysarthria, disorientation, hyperreflexia, tremors, and ataxia. The most important hematologic effect is depression of red blood cell formation.

One human death resulting from accidental ingestion of methyl cellosolve was reported in 1946. This patient was comatose throughout period of observation. Autopsy revealed severe liver and kidney damage and moderate hemorrhagic gastritis.

Cellosolve, butyl cellosolve, and the cellosolve acetates have not produced systemic intoxication in industry. These compounds have been responsible for central nervous system depression, renal damage, and alterations in blood elements and toxicity in certain laboratory animals. See Ethylene Glycol.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

*Cellosolve*, 200 parts per million parts of air by volume or 740 milligrams per cubic meter of air.

*Cellosolve acetate*, 100 parts per million parts of air by volume or 540 milligrams per cubic meter of air.

*Methyl cellosolve*, 25 parts per million parts of air by volume or 80 milligrams per cubic meter of air.

*Methyl cellosolve acetate*, 25 parts per million parts of air by volume or 120 milligrams per cubic meter of air.

*Butyl cellosolve*, 50 parts per million parts of air by volume or 240 milligrams per cubic meter of air.

### Potential Occupational Exposures

Cellophane sealers	Nitrocellulose makers
Cellosolve workers	Oil processors
Cleaning solution makers	Paint makers
Cotton thread makers	Perfume makers
Dope makers	Photographic film makers
Dry cleaners	Printers
Dry cleaning agent makers	Resin makers
Dye makers	Sludge removing agent makers
Enamel makers	Soap makers
Film makers	Stainers
Gum processors	Stain makers
Hydraulic fluid makers	Textile dyers
Insecticide makers	Textile printers
Lacquer makers	Varnish makers
Lacquer thinner makers	Varnish remover makers
Leather makers	Wax processors
Nail polish makers	Wood stain makers

### References

CARPENTER, C. P.; POZZANI, U. C.; WEIL, C. S.; NAIR, J. H., III; KECK, G. A.; SMYTH, H. F., JR.: The toxicity of butyl cellosolve solvent. *A.M.A. Arch. Indust. Health* 14: 114, 1956.

YOUNG, E. G. AND WOOLNER, L. B.: A case of fatal poisoning from 2-methoxyethanol. *J. Ind. Hyg. & Toxicol* 28: 267, 1946.

ZAVON, M. R.: Methyl cellosolve intoxication. *Am. Indust. Hyg. Assoc. J.* 24: 36, 1963.

### (43) Cement, Portland

### Harmful Effects

*Local* Exposure may produce cement dermatitis which is usually due to primary irritation from alkalinity, hygroscopicity, or abrasive property of

cement. In some cases cement workers have developed an allergic sensitivity to constituents of cement such as hexavalent chromate. It is not unusual for cement dermatitis to be prolonged and to involve covered areas of body.

*Route of Entry* Inhalation of dust.

*Systemic* No systemic manifestations attributable to cement exposure have been reported.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

50 million particles per cubic foot of air.

### *Potential Occupational Exposures*

Asbestos cement pipe makers	Heat insulation makers
Asbestos cement sheet makers	Oil well builders
Asbestos cement shingle makers	Pier builders
Barge builders	Post makers
Brick masons	Reservoir builders
Bridge builders	Road construction workers
Building construction workers	Sidewalk builders
Burial vault builders	Silo builders
Cement insulation makers	Smokestack builders
Cement insulation workers	Sound insulation makers
Cement makers	Stadium builders
Cement pipe makers	Storage tank builders
Cement workers	Swimming pool builders
Concrete runway builders	Tunnel builders
Dam builders	Water pipe makers
Drain tile makers	

### *References*

CALNAN, C.: Cement dermatitis. *J. Occup. Med.* 2: 15, 1960.  
 MORRIS, G. E.: The primary irritant nature of cement. *Arch. Environ. Health* 1: 301, 1960.  
 SANDER, O. A.: Roentgen resurvey of cement workers. *A.M.A. Arch. Indust. Health* 17: 96, 1958.

## (44) Cerium

### *Harmful Effects*

*Local* None reported.

*Route of Entry* Inhalation of dust.

*Systemic* No cases of industrial poisoning have been found in literature. Cerium and its salts have produced alterations in blood elements in certain experimental animals.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Alloy makers	Ink makers
Ammonia makers	Lighter flint makers
Cerium workers	Metal refiners
Enamel makers, vitreous	Phosphor makers
Explosive makers	Photographic illuminant makers
Glass makers	Rocket fuel makers
Glass polish makers	Textile workers

(45) Chlordane. *See Pesticides Section*

(46) Chloride of Lime

*chlorinated lime, bleaching powder*

Chloride of lime is a mixture of calcium chloride, calcium hypochlorite and calcium hydroxide.

*Harmful Effects*

*Local* The powder and its solutions have corrosive action on skin, eyes and mucous membranes and can produce conjunctivitis, blepharitis, corneal ulceration, gingivitis, and contact dermatitis.

*Route of Entry* Inhalation of dust.

*Systemic* Dust is irritating to respiratory tract, and can produce laryngitis and pulmonary edema.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Chloride of lime workers	Sewage treaters
Deodorant makers	Soap bleachers
Disinfectant makers	Straw bleachers
Dyers	Textile bleachers
Laundry workers	Textile printers
Oil bleachers	Water treaters
Organic chemical synthesizers	Wood pulp bleachers
Paper makers	

## (47) Chlorinated Benzenes

*Chlorobenzene*: phenyl chloride, monochlorobenzene, chlorobenzol

*o-Dichlorobenzene*: 1,2-dichlorobenzene

*p-Dichlorobenzene*: 1,4-dichlorobenzene

*Trichlorobenzenes*: 1,2,4-trichlorobenzene; 1,3,5-trichlorobenzene

*Hexachlorobenzene*: perchlorobenzene

### *Harmful Effects*

*Local* Chlorinated benzenes are irritating to skin, conjunctiva, and mucous membranes of upper respiratory tract.

*Routes of Entry* Inhalation of vapor, percutaneous absorption of liquid. Percutaneous route is of little importance when contact is with solid p-dichlorobenzene.

*Systemic* Studies of industrial populations exposed to o-dichlorobenzene and p-dichlorobenzene reveal no significant systemic effects. Liver injury and cataracts have been reported with high exposures to certain of these compounds.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

*Chlorobenzene*, 75 parts per million parts of air by volume or 350 milligrams per cubic meter of air.

*o-Dichlorobenzene*, 50 parts per million parts of air by volume or 300 milligrams per cubic meter of air.

*p-Dichlorobenzene*, 75 parts per million parts of air by volume or 450 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

#### *Chlorobenzene*

Aniline makers

Carbolic acid makers

Cellulose acetate workers

Chlorobenzene workers

DDT makers

Drug makers

Dry cleaners

Dyers

Ethyl cellulose workers

Heat transfer workers

Ink makers

Lacquerers

Lacquer makers

Organic chemical synthesizers

Paint workers

Perfume makers

Picric acid makers

Resin makers

Rubber makers

Sulfur dye makers

Varnish makers

#### *o-Dichlorobenzene*

Asphalt makers

Cleaning compound makers

Deodorant makers

o-Dichlorobenzene workers	Varnish makers
Dry cleaners	Varnish remover workers
Dye makers	Wax makers
Fumigant workers	Wood preservative workers
Greasemakers	Wool processors
Gum makers	<i>p-Dichlorobenzene</i>
Heat transfer workers	Deodorant makers
Hide processors	p-Dichlorobenzene workers
Insecticide workers	Disinfectant workers
Lacquerers	Drug makers
Lacquer workers	Dye makers
Metal degreasers	Insecticide workers
Metal polish makers	Moth ball makers
Organic chemical synthesizers	Soil fumigators
Paint remover workers	<i>Trichlorobenzenes</i>
Paint workers	Dye makers
Polishing compound makers	Electric equipment makers
Resin makers	Heat transfer workers
Rubber makers	Insecticide workers
Solvent workers	Lubricant makers
Stainers	Trichlorobenzene workers
Stain makers	<i>Hexachlorobenzene</i>
Sulfur processors	Fungicide workers
Tannery workers	Hexachlorobenzene workers
Tar makers	Organic chemical synthesizers
Tar remover workers	Seed disinfectors
Termite exterminator workers	

## References

HOLLINGSWORTH, R. L.; ROWE, V. K.; OYEN, F.; HOYLE, H. R., AND SPENCER, H. C.: Toxicity of paradichlorobenzene; determinations on experimental animals and human subjects. *A.M.A. Arch. Indust. Health* 14: 138, 1956.

HOLLINGSWORTH, R. L.; ROWE, V. K.; OYEN, F.; TORKELSON, T. R., AND ADAMS, E. M.: Toxicity of o-dichlorobenzene; studies on animals and industrial experience. *A.M.A. Arch. Indust. Health* 17: 180, 1958.

## (48) Chlorinated Diphenyls and Naphthalenes

### Harmful Effects

*Local* Prolonged contact with fume or the cold wax leads on exposed skin to comedones, sebaceous cysts and pustules, known as chloracne.

*Routes of Entry* Inhalation of fume or vapor; percutaneous absorption of liquid.

*Systemic* Acute or chronic exposure can produce varying degrees of liver damage depending on amount of chlorine in compound and preexisting

state of liver. Symptoms include jaundice, anorexia, nausea, indigestion, abdominal pains, and edema. Death from acute yellow atrophy of liver has occurred.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

*Chlorodiphenyl (42 percent chlorine)*, 1 milligram per cubic meter of air.

Should be reduced when also absorbed percutaneously.

*Chlorodiphenyl (54 percent chlorine)*, 0.5 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Chlorinated diphenyl oxide*, 0.5 milligram per cubic meter of air.

*Pentachloronaphthalene*, 0.5 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Trichloronaphthalene*, 5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Aniline dye makers	Mineral oil processors
Cable coaters	Moisture proofers
Carbon removers	Paint makers
Chlorinated diphenyl workers	Paper treaters
Condenser impregnators	Petroleum refinery workers
Crankcase oil additive makers	Plasticizer makers
Dye makers	Plastic makers
Electric equipment makers	Rayon makers
Electricians	Resin makers
Electroplaters	Rubber workers
Flameproofers	Solvent workers
Gum processors	Stainers
Heat transfer workers	Stain makers
Herbicide workers	Textile flameproofers
Ink makers	Transformer workers
Insecticide workers	Upper cylinder oil makers
Insect proofers	Varnish makers
Lacquerers	Vegetable oil processors
Lacquer makers	Wax makers
Light fixture makers	Wire coaters
Machinists	Wood preservers
Metal degreasers	

### *Reference*

MEIGS, J. W.; ALBOM, J. J.; AND KARTIN, B. L.: Chloracne from an unusual exposure to arochlor. *J. Am. Med. Assoc.* 154: 1417, 1954

## (49) Chlorine

*Harmful Effects*

*Local* Extreme irritation of skin, eyes, and mucous membranes; corrosion of teeth.

*Route of Entry* Inhalation of gas.

*Systemic* Acute respiratory distress including cough, hemoptysis, chest pain, dyspnea, and cyanosis. Later, tracheobronchitis, bronchopneumonia, and pulmonary edema may supervene.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

1 part per million parts of air by volume or 3 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Aerosol propellant makers	Laundry workers
Alkali salt makers	Methyl chloride makers
Aluminum purifiers	Paper bleachers
Benzene hexachloride makers	Petroleum refinery workers
Bleachers	Phosgene makers
Bleaching powder makers	Photographic workers
Bromine makers	Pulp bleachers
Broom makers	Rayon makers
Carpet makers	Refrigerant makers
Chemical synthesizers	Rubber makers
Chloride of lime makers	Sewage treaters
Chlorinated solvent makers	Silver extractors
Chlorine workers	Sodium hydroxide makers
Color makers	Submarine workers
DDT makers	Sugar refiners
Disinfectant makers	Sulfur chloride makers
Dye makers	Swimming pool maintenance
Ethylene glycol makers	workers
Ethylene oxide makers	Tetraethyl lead makers
Flour bleachers	Textile bleachers
Freon makers	Tin recovery workers
Gasoline additive workers	Toxaphene makers
Gold extractors	Vinyl chloride makers
Ink makers	Vinylidene chloride makers
Iodine makers	Water treaters
Iron detinners	Zinc chloride makers
Iron dezinkers	

*References*

CHASIS, H.; ZAPP, J. A.; BANNON, J. H.; WHITTENBERGER, J. L.; HELM, J.; DOHENY, J. J., AND MACLEOD, C. M.: Chlorine accident in Brooklyn. *Occup. Med.* 4: 152, 1947.

JOYNER, R. E., AND DURIEL, E. G.: Accidental liquid chlorine spill in a rural community. *J. Occup. Med.* 4: 152, 1962.

(50) **Chloroprene***chlorobutadiene**Harmful Effects*

*Local* May cause contact dermatitis from primary skin irritation; may produce mucous membrane irritation and temporary hair loss.

*Routes of Entry* Percutaneous absorption; inhalation of vapor.

*Systemic* Chloroprene may cause lung irritation, liver and kidney damage, and central nervous system depression.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

25 parts per million parts of air by volume or 90 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Chloroprene workers

Rubber makers, neoprene

*References*

RITTER, W. L. AND CARTER, A. S.: Hair loss in neoprene manufacture. *J. Indust. Hyg. & Toxicol.* 30: 192, 1948.

VON OETTINGEN, W. F.; HUEPER, W. C.; DEICHMANN-GRUEBLER, W., AND WILEY, F. H.: 2-Chloro-butadiene (chloroprene); its toxicity and pathology, and the mechanism of its action. *J. Indust. Hyg. & Toxicol.* 18: 240, 1936.

(51) **Chromium Compounds**

Chromium compounds include chromic acid (chromic trioxide), chromates, and bichromates.

*Harmful Effects*

*Local* Contact with chromates or chromic acid can produce small, painless cutaneous ulcers as well as dermatitis from primary irritation or allergic hypersensitivity. Cutaneous allergy is not uncommon from hexavalent chromium compounds but is extremely rare from trivalent chromium compounds. Yellowish discoloration of teeth and tongue; perforation of nasal septum; conjunctivitis.

*Route of Entry* Inhalation of dust or mist.

*Systemic* Allergic bronchial asthma from chromium trioxide fume. Bronchogenic carcinoma has occurred at an abnormally high rate among chromate workers. The carcinogenic form of chromium has not been determined.

### *Special Diagnostic Test*

Determination of chromium in blood and urine. *See* Division of Occupational Health, 1953.

### *Recommended Threshold Limit*

Chromic acid and chromates (as CrO<sub>3</sub>), 0.1 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Abrasive makers	Electroplaters
Acetylene purifiers	Enameler workers
Adhesive workers	Explosive makers
Airplane sprayers	Fat purifiers
Alizarin makers	Fireworks makers
Alloy makers	Fly paper makers
Aluminum anodizers	Furniture polishers
Aniline black makers	Fur processors
Anodizers	Glass fiber makers
Battery makers, dry	Glass frosters
Biologists	Glass makers
Blue print makers	Glass makers, colored
Boiler scalers	Glue makers
Candle makers, colored	Histology technicians
Cement workers	Ink makers
Ceramic workers	Jewelers
Chromate workers	Laboratory workers, chemical
Chrome alloy workers	Leather finishers
Chrome alum workers	Linoleum workers
Chromium platers	Lithographers
Chromium workers	Magnesium treaters
Color makers	Match makers
Copper etchers	Metal cleaners
Copper plate strippers	Metal cutters
Corrosion inhibitor workers	Metal etchers
Crayon makers, colored	Metal treaters
Diesel locomotive repairmen	Milk preservers
Drug makers	Mordanters
Dry color makers	Oil drillers
Dye makers	Oil purifiers
Dyers	Organic chemical synthesizers

Painters	Refractory brick makers
Paint makers	Rubber makers
Palm oil bleachers	Rust inhibitor workers
Paper dyers	Shingle makers
Paper waterproofers	Silk screen makers
Pencil makers, colored	Smokeless powder makers
Perfume makers	Soap makers
Photoengravers	Sponge bleachers
Photographers	Stainless steel workers
Photographic chemical makers	Tanners
Pigment makers	Textile dyers
Platinum polishers	Textile mordanters
Porcelain decorators	Textile printers
Pottery frosters	Textile waterproofers
Pottery glaze makers	Wallpaper printers
Pottery glazers	Wax bleachers
Printers	Wax ornament workers
Printing ink workers	Welders
Process engravers	Wood preservative workers
Pyrotechnic workers	Wood stainers
Railroad engineers	Wood stain makers

### References

BAETJER, A. M.: Pulmonary carcinoma in chromate workers. 1, A review of the literature and report of cases. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 2: 487, 1950.

BERNHARDT, H. J.: Chromate dermatitis; its natural history and treatment. *A.M.A. Arch. Dermat.* 76: 13, 1957.

DENTON, C. R.; KEENAN, R. G., AND BIRMINGHAM, D. J.: The chromium content of cement and its significance in cement dermatitis. *J. Invest. Dermat.* 23: 189, 1954.

DIVISION OF OCCUPATIONAL HEALTH, PUBLIC HEALTH SERVICE: Health of workers in chromate producing industry. Pub. Health Service Pub. No. 192. U.S. Government Printing Office, Washington, D.C., 1953.

MANCUSO, T. F. AND HUEPER, W. C.: Occupational cancer and other health hazards in a chromate plant; a medical appraisal. 1, Lung cancer in chromate workers. *Indust. Med. & Surg.* 20: 358, 1951.

MANCUSO, T. F.: Occupational cancer and other health hazards in a chromate plant; a medical appraisal. 2, Clinical and toxicologic aspects. *Indust. Med. & Surg.* 20: 393, 1951.

WINSTON, J. R. AND WALSH, E. N.: Chromate dermatitis in railroad employees working with diesel locomotives. *J. Am. Med. Assoc.* 147: 1133, 1951.

### (52) Coal Tar and Fractions

Coal tar, derived from destructive distillation of coal during manufacture of coke and illuminating gas, can be divided by distillation into several crude fractions: crude naphtha, creosote oil, anthracene oil and pitch. For specific constituents see Acridine, Benzene, Cresol, Naphtha, Naphthalenes (Chlorinated Diphenyls and Naphthalenes), Phenol, and Toluene.

## *Harmful Effects*

**Local** Photosensitization may occur and is manifested by erythema, edema, burning, and subsequent hyperpigmentation of exposed areas. Other cutaneous effects include folliculitis, acne, and comedones; keratoses, papillomas, and squamous cell epitheliomas following years of exposure; contact dermatitis from either primary irritation or allergic hypersensitivity; and conjunctivitis.

**Route of Entry** Inhalation of dust or vapor.

**Systemic** Overexposure to vapor produces anorexia, nausea, vomiting, and cough. Bronchogenic carcinoma has been suspected from inhalation of coal tar vapors and dust in Great Britain, Canada, and Japan.

## *Special Diagnostic Tests*

Examination of skin under Wood's light for fluorescence of residual tar.

Histologic examination of skin biopsy for malignancy.

## *Recommended Threshold Limit*

Not established.

## *Potential Occupational Exposures*

Artificial stone makers	Flue cleaners
Asbestos goods workers	Fuel pitch workers
Asphalt workers	Furnace men
Battery box makers	Gas house workers
Battery workers, dry	Glass blowers
Boat builders	Impregnated felt makers
Brick masons	Insecticide bomb makers
Brick pressers	Insulation board makers
Brickyard workers	Insulators
Briquette makers	Lens grinders
Brush makers	Linemen
Cable makers	Miners
Carpenters	Painters
Coal tar still cleaners	Paper conduit makers
Coal tar workers	Pavers
Coke oven workers	Pipeline workers
Corkstone makers	Pipe pressers
Creosoters	Pitch workers
Diesel engine engineers	Railroad track workers
Electric equipment makers	Riveters
Electricians	Road workers
Electrode makers	Roofers
Electrometallurgic workers	Roofing paper workers
Farmers	Rope makers
Fishermen	Rubber workers

Shingle makers	Tar paint makers
Shipyard workers	Tile pressers
Soap makers	Waterproof concrete workers
Smokeless fuel makers	Waterproofers
Stokers	Wood preservers

### References

DOLL, R.: Occupational lung cancer; a review. *Brit. J. Indust. Med.* 16: 181, 1959.  
 FISHER, R. E. W.: Skin cancer in tar workers. *Trans. Assoc. Indust. Med. Officers* 3: 315, 1954.

## (53) Cobalt and Compounds

### Harmful Effects

*Local* Metallic cobalt dust and cobalt salts may produce allergic contact dermatitis. Dust of certain cobalt ores may irritate cornea.

*Route of Entry* Inhalation of dust.

*Systemic* Bronchial asthma from inhalation of cobalt dust has been suspected. Inhalation of anyhydrous cobalt acetate dust has produced gastrointestinal irritation. A pneumoconiosis of tungsten carbide workers, manifested by cough and dyspnea, has been reported.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

(Cobalt) 0.5 milligram per cubic meter of air.

### Potential Occupational Exposures

Acetic acid makers	Electroplaters
Actors	Enamelters
Alloy makers	Ethyl acrylate makers
Alnico magnet makers	Fertilizer workers
Ammonia mask makers	Frit workers
Barometer makers	Gas mask makers
Bright platers	Gasoline blenders
Catalyst workers	Glass colorers
Cement makers	Glaze workers
Cemented (tungsten) carbide workers	High speed tool steel workers
Ceramic workers	Hygrometer makers
Cermet makers	Ink makers, sympathetic
Cobalt soap makers	Iron cobalt-platers
Cobalt workers	Lacquer dryer makers
Cosmetic makers	Lacquer makers
Drug makers	Lamp filament makers
	Magnet steel workers

Metallurgists	Paint makers
Mineral feed makers	Phthalic anhydride makers
Moisture indicator makers	Polyester resin workers
Nickel workers	Porcelain colorers
Nuclear technologists	Porcelain enamel workers
Oilcloth color workers	Pottery glaziers
Oil dryer makers	Rubber colorers
Oil hydrogenators	Varnish dryer makers
Oil pigment makers	Varnish makers
Paint dryer makers	

### References

FAIRHALL, L. T.; KEENAN, R. G., AND BRINTON, H. P.: Cobalt and the dust environment of the cemented tungsten carbide industry. *Pub. Health Rep.* 64: 485, 1949.

MILLER, C. W.; DAVIS, M. W.; GOLDMAN, A., AND WYATT, J. P.: Pneumoconiosis in the tungsten-carbide tool industry. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 453, 1953.

## (54) Copper and Compounds

### Harmful Effects

*Local* The following copper salts have been reported to be skin and mucous membrane irritants: copper arsenite, copper cyanide, copper fluoride, copper naphthenate, copper oxide, and copper sulfate.

*Route of Entry* Inhalation of dust or fume.

*Systemic* Inhalation of copper fumes has been reported to produce signs and symptoms of metal fume fever. These include chills, transient fever, nausea, thirst, and exhaustion. Prolonged inhalation of copper salts may cause perforation of nasal septum.

### Special Diagnostic Tests

Examination of blood and urine for excessive amounts of copper. See Stewart and Stolman, 1960.

### Recommended Threshold Limits

(Tentative) Copper (fume), 0.1 milligram per cubic meter of air; copper (dusts, mists), 1 milligram per cubic meter of air.

### Potential Occupational Exposures

Antifouling paint makers	Copper smelters
Asphalt makers	Coppersmiths
Battery makers	Copper workers
Brass founders	Electroplaters
Canvas preservative workers	Enamel workers
Copper founders	Flotation workers
Copper platers	Fungicide workers
Copper refiners	Gem colorers

Glass makers	Rayon makers
Glue makers	Refrigerator makers
Hair dye workers	Rope preservative workers
Hide preservative workers	Rubber makers
Ink makers	Solderers
Insecticide workers	Steel makers
Lithographers	Tanners
Organic chemical synthesizers	Textile dyers
Paint makers	Textile makers
Petroleum refinery workers	Wallpaper makers
Pigment makers	Water treaters
Propeller polishers	Wood preservative workers
Railroad tie preservative workers	

### References

DAVENPORT, S. J.: Review of literature on health hazards of metals. 1, Copper. Bureau of Mines Information Circular 7666. U.S. Department of Interior, Washington, D.C., 1953.

STEWART, C. P. AND STOLMAN, A.: *Toxicology; Mechanisms and Analytical Methods*. Vol. 1. Academic Press, New York, 1960.

### (55) Cresol

*cresylic acid, cresylol, hydroxytoluene, methyl phenol, oxytoluene, tricresol*

Cresol is a mixture of the three isomeric cresols: ortho-, meta-, and para-.

### Harmful Effects

*Local* Cresol, a potent primary irritant, has a corrosive action on skin and mucous membranes. Intense irritation is produced upon contact with eye.

*Routes of Entry* Inhalation or percutaneous absorption of liquid or vapor.

*Systemic* Inhalation of vapor may cause pulmonary edema. Severe poisoning is followed by collapse, hypothermia, and death. Nonfatal poisoning may be followed by severe liver and kidney damage which appear after a period of apparent full recovery.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

(All isomers), 5 parts per million parts of air by volume or 22 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

## Potential Occupational Exposures

Coal tar workers	Oil additive makers
Cresol soap makers	Paint remover makers
Cresol workers	Paint removers
Cresylic acid makers	Perfume makers
Deodorant workers	Photographic developer workers
Disinfectant makers	Pitch workers
Disinfectors	Resin makers
Dye makers	Roofers
Enamel makers	Rubber makers
Explosive workers	Scouring compound makers
Flotation agent makers	Stainers
Flotation workers	Stain makers
Foundry workers	Surfactant makers
Glue workers	Tar distillery workers
Ink makers	Textile sizers
Ink remover makers	Varnish remover makers
Ink removers	Varnish removers
Insecticide workers	Veterinarians
Insulation enamel workers	Wool scourers

## Reference

FAIRHALL, L. T.: *Industrial Toxicology*. 2nd ed. Williams & Wilkins Co., Baltimore, 1957.

(56) Cyanides. *See* Hydrogen Cyanide

(57) Cyclohexane. *See* Cycloparaffins

(58) Cyclohexene. *See* Cycloparaffins

(59) Cycloparaffins

*cycloalkanes*; included in this classification are cyclohexane or hexahydrobenzene, and cyclohexene or 1,2,3,4-tetrahydrobenzene

## Harmful Effects

*Local* Eye irritation and dry, scaly, fissured dermatitis can be produced by contact with liquid.

*Route of Entry* Inhalation of vapor.

*Systemic* Cycloparaffins are weakly narcotic; in high concentrations may produce headache, dizziness, nausea, vomiting, and unconsciousness.

## Special Diagnostic Test

None.

*Recommended Threshold Limit*

*Cyclohexane*, 400 parts per million parts of air by volume or 1,400 milligrams per cubic meter of air.

*Cyclohexene*, 400 parts per million parts of air by volume or 1,350 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Adipic acid makers	Organic chemical synthesizers
Benzene makers	Paint remover makers
Bitumen processors	Paint removers
Cellulose plastic makers	Perfume makers
Cycloparaffin workers	Plastic molders
Essential oil extractors	Resin makers
Fat processors	Rubber makers
Fungicide makers	Solid fuel makers, camp stove
Lacquerers	Varnish remover makers
Lacquer makers	Varnish removers
Maleic acid makers	
Nylon makers	Wax makers
Oil processors	

(60) DDT. *See* Pesticides Section

(61) Diacetone Alcohol

*diacetone, diacetonyl alcohol, dimethylacetonyl carbinol*

*Harmful Effects*

*Local* Irritation of eyes, nose, and throat by high vapor concentrations.

*Route of Entry* Inhalation of vapor.

*Systemic* Effects have not been described for man. In experimental animals there may be somnolence, narcosis, hypotension, transient anemia, and kidney damage.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

50 parts per million parts of air by volume or 240 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Animal tissue preservers	Cellulose acetate workers
Antifreeze makers	Cellulose ester lacquer makers
Celluloid cement makers	Cellulose nitrate workers

Diacetone alcohol workers	Oil processors
Dope workers	Paint remover makers
Drug makers	Paint removers
Dye makers	Paper coaters
Fat processors	Printers
Garage mechanics	Rayon makers
Gold leaf makers	Resin makers
Hydraulic brake fluid makers	Solvent workers
Ink makers, quick drying	Stainers
Lacquerers	Stain makers
Lacquer makers	Tar processors
Leather makers	Textile workers
Metal cleaners	Wax makers
Nitrocellulose workers	Wood preservative workers

### *Reference*

VON OETTINGEN, W. F.: The aliphatic alcohols; their toxicity and potential dangers in relation to their chemical constitution and their fate in metabolism. Pub. Health Bull. No. 281. U.S. Government Printing Office, Washington, D.C., 1943.

### (62) 1,2-Dichloroethylene

*acetylene dichloride*

#### *Harmful Effects*

*Local* The solvent can act as primary irritant producing contact dermatitis. Vapor can cause irritation of mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of vapor.

*Systemic* Transient narcosis can result from inhalation of vapor. No chronic toxicity in man has been reported.

#### *Special Diagnostic Test*

None.

#### *Recommended Threshold Limit*

200 parts per million parts of air by volume or 790 milligrams per cubic meter of air.

#### *Potential Occupational Exposures*

Camphor processors	Dye makers
Carbolic acid processors	Fat processors
Cellulose acetate workers	Gum processors
Dichloroethylene workers	Lacquerers
Drug makers	Lacquer makers
Dry cleaners	Oil processors

Organic chemical synthesizers	Rubber makers
Perfume makers	Shellac processors
Plastic makers	Solvent workers
Resin makers	Wax makers

### Reference

MCBIRNEY, R. S.: Trichloroethylene and dichloroethylene poisoning. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 10: 130, 1954.

### (63) Dichloroethyl Ether

*dichloroether, dichloroethyl oxide*

### Harmful Effects

*Local* Irritation of conjunctiva and mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of vapor.

*Systemic* No cases of industrial systemic intoxication have been reported. Animal studies indicate that vapor is an intense respiratory tract irritant causing pulmonary edema.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

15 parts per million parts of air by volume or 90 milligrams per cubic meter of air.

### Potential Occupational Exposures

Degreasers	Paint makers
Dichloroethyl ether workers	Pectin processors
Dry cleaners	Resin makers
Ethyl cellulose processors	Soap makers
Fat processors	Soil fumigant workers
Finish remover makers	Soil fumigators
Fulling compound makers	Solvent workers
Gum processors	Stain removers
Lacquer makers	Tar processors
Oil processors	Textile scourers
Oil purifiers	Varnish workers
Organic chemical synthesizers	

### Reference

SCHRENK, H. H.; PATTY, F. A., AND YANT, W. P.: Acute response of guinea pigs to vapors of some new commercial organic compounds. 7, Dichloroethyl ether. *Pub. Health Rep.* 48: 1389, 1933. Reprint no. 1602.

(64) Dieldrin. *See* Pesticides Section

(65) Dimethylformamide

*the "universal organic solvent", DMF*

### *Harmful Effects*

*Local* Highly irritating to skin, eyes, and mucous membranes.

*Route of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* DMF causes gastric irritation with anorexia, nausea, vomiting, epigastric burning, and abdominal pain and tenderness; severe liver and kidney damage reported in experimentally exposed animals.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

20 parts per million parts of air by volume or 60 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Acetylene purifiers	Organic chemical synthesizers
Butadiene makers	Petroleum refinery workers
Dimethylformamide workers	Resin makers
Drug makers	Solvent workers
Dye makers	Synthetic fiber makers
Lubricating oil extractors	

### *Reference*

MASSMANN, W.: Toxicological investigations on dimethylformamide. *Brit. J. Indust. Med.* 13: 51, 1956.

(66) Dimethylhydrazine

*UDMH, 1,1-dimethylhydrazine, asymmetrical dimethylhydrazine*

### *Harmful Effects*

*Local* Liquid is low grade primary irritant of skin. Liquid and vapor are irritating to eyes.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Systemic effects include delayed gastrointestinal irritation, hemolytic anemia, and possible liver damage. Vapor produces irritation of respiratory tract. On the basis of animal experiments, UDMH may cause convulsions and kidney damage.

### *Special Diagnostic Test*

None.

*Recommended Threshold Limit*

0.5 part per million parts of air by volume or 1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Jet fuel handlers	Photographic developer makers
Jet fuel makers	Rocket fuel handlers
Organic chemical synthesizers	Rocket fuel makers

*References*

JACOBSON, K. H.; CLEM, J. H.; WHEELWRIGHT, H. J., JR.; RINEHART, W. E., AND MAYES, N.: The acute toxicity of the vapors of some methylated hydrazine derivatives. *A.M.A. Arch. Indust. Health* 12: 609, 1955.

JACOBSON, K. H.: Industrial hygiene aspects of liquid propellants. *Transactions, 22nd annual meeting, American Conference of Governmental Industrial Hygienists, 1960.* Sec.-Treas., 1014 Broadway, Cincinnati 2, Ohio.

OFFICE OF DIRECTOR, DEFENSE RESEARCH AND ENGINEERING, DEPARTMENT OF DEFENSE: *The Handling and Storage of Liquid Propellants.* U.S. Government Printing Office, Washington, D.C., 1961.

SHOOK, B. S., SR., AND COWART, O. H.: Health hazards associated with unsymmetrical dimethylhydrazine. *Indust. Med. & Surg.* 26: 333, 1957.

## (67) Dimethyl Sulfate

*methyl sulfate*

*Harmful Effects*

*Local* Liquid or vapor produces vesication and analgesia on contact with skin. Analgesia may persist for several months thereafter. Mucous membranes are irritated by liquid or vapor, with production of conjunctivitis, lacrimation, corneal ulcerations, rhinitis, edema of mucosa of mouth and throat, dysphagia, sore throat, and hoarseness. Irritation of skin and mucous membranes may be delayed in appearance.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Irritation of lungs produces bronchitis, pneumonitis, and pulmonary edema. Absorption is followed by cerebral edema and central nervous system effects such as drowsiness, paralysis, convulsions, delirium, and coma. Absorption may also produce liver and kidney damage.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

1 part per million parts of air by volume or 5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

## Potential Occupational Exposures

Amine makers	Methylation workers
Color makers	Organic chemical synthesizers
Drug makers	Perfume makers
Dye makers	Phenol derivative makers

## References

GAULTIER, M.; FOURNIER, E.; GERVAIS, P.; GORCEIX, A., AND EFTHYMIOU, T.: Two cases of methyl sulfate poisoning. *Arch. Mal. Prof.* 21: 744, 1960. (*Indust. Hyg. Digest*, Abst. No. 987, October 1961.)

HASWELL, R. W.: Dimethyl sulfate poisoning by inhalation. *J. Occup. Med.* 2: 454, 1960.

LITTLER, T. R. AND MCCONNELL, R. B.: Dimethyl sulfate poisoning. *Brit. J. Indust. Med.* 12: 54, 1955.

## (68) Dinitrobenzene

*Dinitrobenzol; meta-, ortho- and para-isomers*

## Harmful Effects

*Local* Dinitrobenzene is a primary skin irritant.

*Routes of Entry* Percutaneous absorption of liquid; inhalation of vapor.

*Systemic* Systemic effects are similar to Nitrobenzene (which see).

## Special Diagnostic Tests

Analysis of urine for dinitrobenzene and blood for methemoglobin. See Von Oettingen, 1958, and Stewart and Stolman, 1961.

## Recommended Threshold Limit

1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

## Potential Occupational Exposures

Celluloid makers	Explosive users
Dinitrobenzene workers	Organic chemical synthesizers
Dye makers	Plastic makers
Explosive makers	

## References

BERITIC, T.: Two cases of meta-dinitrobenzene poisoning with unequal clinical response. *Brit. J. Indust. Med.* 13: 114, 1956.

STEWART, C. P. AND STOLMAN, A.: *Toxicology; Mechanisms and Analytical Methods*. Vol. 2. Academic Press, New York, 1961.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (69) Dinitrophenol

Of the 6 isomers the 2,4- is the most toxic.

*Harmful Effects*

*Local* Yellow staining of skin. Eczematous dermatitis due to either primary irritation or allergic hypersensitivity. Exfoliative dermatitis has occurred.

*Routes of Entry* Percutaneous absorption from dust; inhalation of dust; to a lesser extent, ingestion.

*Systemic* Dinitrophenol blocks oxidative phosphorylation and thereby stimulates basal metabolism with resultant effects of anorexia, nausea, vomiting, sweating, thirst, dyspnea, excitement, tachycardia, and fever. Acidosis may develop. Central nervous system effects are those of stimulation followed by depression. There may be cataract formation, kidney or liver damage. Death may result from overwhelming exposure.

*Special Diagnostic Test*

Detection of dinitrophenol and aminonitrophenol in urine. See Von Oettingen, 1958.

*Recommended Threshold Limit*

0.2 milligram per cubic meter of air. See American Industrial Hygiene Association, 1958.

*Potential Occupational Exposures*

Diaminophenol makers	Indicator makers, chemical
Dinitrophenol workers	Organic chemical synthesizers
Dye makers	Photographic developer makers
Explosive workers	Wood preservative workers
Herbicide workers	

*References*

AMERICAN INDUSTRIAL HYGIENE ASSOCIATION: 2,4-Dinitrophenol. *Hygienic Guide Series*. The Association, Detroit, 1958. Recommended threshold limit: 0.2 milligram per cubic meter of air.

GISCLARD, J. B. AND WOODWARD, M. M.: 2,4-Dinitrophenol poisoning; a case report. *J. Indust. Hyg. & Toxicol.* 28: 47, 1946.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (70) Dinitrotoluene

*dinitrotoluol, DNT*

*Harmful Effects*

*Local* Contact may produce allergic hypersensitization.

*Routes of Entry* Percutaneous absorption of liquid; inhalation of vapor.  
*Systemic* Symptoms and signs are similar to intoxication from trinitrotoluene. *See* Trinitrotoluene.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

1.5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### Potential Occupational Exposures

Dinitrotoluene workers

Explosive workers

Dye makers

Organic chemical synthesizers

### (71) Dioxane

*1,4-diethylene dioxide, diethylene ether*

### Harmful Effects

*Local* Irritation of eyes, nose and throat.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Severe gastric symptoms. Liver necrosis and nephritis.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

100 parts per million parts of air by volume or 360 milligrams per cubic meter of air.

### Potential Occupational Exposures

Adhesive workers

Lacquerers

Cellulose acetate workers

Lacquer makers

Cellulose ester workers

Metal cleaners

Cement workers

Oil processors

Cosmetic makers

Painters

Degreasers

Paint makers

Deodorant makers

Paint removers

Detergent workers

Paint remover workers

Dioxane workers

Plastic makers

Dye makers

Polish makers

Emulsion makers

Printers

Fat processors

Resin makers

Fumigant workers

Shoe cream makers

Glue makers

Solvent workers

Histology technicians

Stainers

Stain makers  
Textile makers  
Varnish makers

Varnish remover makers  
Varnish removers

### References

JOHNSTONE, R. T.: Death due to dioxane? *A.M.A. Arch. Indust. Health* 20: 445, 1959.  
LEHMANN, K. B. AND FLURY, F. (EDITORS); KING, E. AND SMYTH, H. F., JR. (TRANSLATORS): *Toxicology and Hygiene of Industrial Solvents*. Williams & Wilkins Co., Baltimore, 1943.

### (72) Epichlorohydrin *epi, chloropropylene oxide*

#### Harmful Effects

**Local** Liquid and vapor are highly irritating to skin, eyes, and upper respiratory tract. Cutaneous burns may be delayed in appearance. Allergic eczematous contact dermatitis occurs occasionally.

**Route of Entry** Inhalation of vapor.

**Systemic** On the basis of animal experiments, epichlorohydrin is highly irritating to lungs, and may damage liver and kidneys. No cases of pulmonary injury or systemic intoxication have been reported in man.

#### Special Diagnostic Test

None.

#### Recommended Threshold Limit

(Tentative) 5 parts per million parts of air by volume.

#### Potential Occupational Exposures

Cellulose ether workers	Lacquer makers
Epichlorohydrin workers	Nail enamel makers
Epoxy resin makers	Organic chemical synthesizers
Glycerol derivative makers	Painters
Glycerophosphoric acid makers	Paint makers
Glycidol derivative makers	Solvent workers
Gum processors	Varnish makers
Lacquerers	

#### Reference

ANON.: Epichlorohydrin. Toxicity Data Sheet, Industrial Hygiene Bulletin. Shell Chemical Corp., New York, 1959.

### (73) Ethyl Acetate *acetic ether, vinegar naphtha*

#### Harmful Effects

**Local** Vapor may produce irritation of eyes, nose and throat. Concentrated solutions are capable of causing skin irritation. In rare instances, dermatitis from hypersensitivity to ethyl acetate may be encountered.

*Route of Entry* Inhalation of vapor.

*Systemic* Exhibits narcotic action through central nervous system depression. Prolonged inhalation may produce acute pulmonary edema.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

400 parts per million parts of air by volume or 1,400 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Bristle makers	Leather makers
Confection makers	Nitrocellulose makers
Denatured alcohol makers	Organic chemical synthesizers
Dope processors	Perfume makers
Drug makers	Photographic film makers
Ethyl acetate workers	Rayon makers
Explosive makers	Resin makers
Flavoring makers	Smokeless powder makers
Fruit essence makers	Solvent workers
Horsehair makers	Stainers
Ink makers	Stain makers
Lacquerers	Varnish makers
Lacquer makers	

### *Reference*

VON OETTINGEN, W. F.: The aliphatic acids and their esters: toxicity and potential dangers; the saturated monobasic aliphatic acids and their esters. *A.M.A. Arch. Indust. Health* 21: 28, 1960.

### (74) Ethyl Alcohol

*ethanol, grain alcohol, ethyl hydroxide*

### *Harmful Effects*

*Local* Irritant to eyes and mucous membranes. Repeated contact can produce dry, scaly, and fissured dermatitis.

*Route of Entry* Inhalation of vapor.

*Systemic* When inhaled in very high concentrations, a mild degree of alcoholic intoxication may be produced.

### *Special Diagnostic Tests*

Analysis of blood and urine for alcohol. See Gonzales et al., 1954, and Stewart et al., 1960.

*Recommended Threshold Limit*

1,000 parts per million parts of air by volume or 1,900 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Acetaldehyde makers	Histology technicians
Acetic anhydride makers	Ink makers
Antifreeze makers	Motor fuel blenders
Beverage makers	Organic chemical synthesizers
Cleaning compound makers	Rocket fuel handlers
Cosmetic makers	Rocket fuel makers
Denatured alcohol makers	Rubber makers
Detergent makers	Shellac processors
Disinfectant makers	Solvent workers
Distillers	Stainers
Drug makers	Stain makers
Dye makers	Thermometer makers, vapor pressure
Ethyl alcohol workers	Varnish makers
Explosive makers	

*References*

GONZALES, T. A.; VANCE, M.; HELPERN, M., AND UMBERGER, C. J.: *Legal Medicine; Pathology and Toxicology*. 2nd ed. Appleton-Century-Crofts, New York, 1954. Ch. 46.

HENSON, E. V.: The toxicology of some aliphatic alcohols; part 2. *J. Occup. Med.* 2: 497, 1960.

STEWART, R. D.; TORKELSON, T. R.; HAKE, C. L., AND ERLEY, D. S.: Infrared analysis of carbon tetrachloride and ethanol in blood. *J. Lab. & Clin. Med.* 56: 148, 1960.

(75) Ethylbenzene

*ethylbenzol, phenylethane*

*Harmful Effects*

*Local* Exposure to liquid or vapor may produce primary irritation of skin, eyes, and mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of vapor. Percutaneous absorption of liquid resulting in systemic toxicity is not likely to occur through intact skin.

*Systemic* No systemic effects from industrial exposures have been reported. In human experimental studies, dizziness was produced with exposure to 2,000 parts per million parts of air after 6 minutes.

*Special Diagnostic Test*

Analysis of urine for hippuric acid. See Gerarde, 1960.

*Recommended Threshold Limit*

200 parts per million parts of air by volume or 870 milligrams per cubic meter of air.

## Potential Occupational Exposures

Ethylbenzene workers	Organic chemical synthesizers
Lacquerers	Resin makers
Lacquer makers	Solvent workers
Motor fuel makers	Styrene makers

## Reference

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons*. Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

(76) Ethyl Bromide (Bromoethane). *See* Bromine and Compounds

### (77) Ethyl Chloride

*monochloroethane, hydrochloric ether, chloroethane*

## Harmful Effects

*Local* Rapid evaporation from skin may cause mild frostbite. Both liquid and gas may irritate eyes.

*Route of Entry* Inhalation of gas.

*Systemic* Ethyl chloride is a narcotic and produces headache, dizziness, incoordination, and eventual loss of consciousness. In high concentrations it is toxic to cardiac muscle and kidney.

## Special Diagnostic Test

None.

## Recommended Threshold Limit

1,000 parts per million parts of air by volume or 2,600 milligrams per cubic meter of air.

## Potential Occupational Exposures

Anesthetists	Organic chemical synthesizers
Dentists	Perfume makers
Drug makers	Phosphorus processors
Dye makers	Physicians
Ethylation workers	Refrigeration workers
Ethyl cellulose makers	Resin makers
Ethyl chloride workers	Sulfur processors
Fat processors	Tetraethyl lead makers
Oil processors	Wax makers

## Reference

VON OETTINGEN, W. F.: The halogenated aliphatic, olefinic, cyclic, aromatic, and aliphatic-aromatic hydrocarbons including the halogenated insecticides; their toxicity and potential dangers. Pub. Health Service Pub. No. 414, U.S. Government Printing Office, Washington, D.C., 1955.

## (78) Ethylene Chlorohydrin

*glycol chlorohydrin, 2-chloroethanol**Harmful Effects**Local* High vapor concentrations are irritating to eyes, nose, and throat.*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.*Systemic* Early symptoms are dizziness, nausea, vomiting, and drowsiness. Several hours after exposure there may be severe headache, dyspnea, fatigue, cyanosis, chest pain, shock, coma, and death. There may also be pulmonary edema, and liver and kidney damage.*Special Diagnostic Test*

Analysis of blood and urine for ethylene chlorohydrin. See Ballotta et al., 1953.

*Recommended Threshold Limit*

5 parts per million parts of air by volume or 16 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Cellulose acetate workers	Oil of rose makers
Drug makers	Organic chemical synthesizers
Dye makers	Potato growers
Ethyl cellulose workers	Potato sprouters
Ethylene chlorohydrin workers	Procaine makers
Ethylene glycol makers	Resin workers
Ethylene oxide makers	Textile dyers
Indigo makers	Textile printers
Insecticide makers	Varnish makers
Lacquer makers	

*References*

BALLOTTA, F.; BERTAGNI, P., AND TROISI, F. M.: Acute poisoning caused by ingestion of ethylene chlorohydrin. *Brit. J. Indust. Med.* 10: 161, 1953.

BUSH, A. F.; ABRAMS, H. K., AND BROWN, H. V.: Fatality and illness caused by ethylene chlorohydrin in an agricultural occupation. *J. Indust. Hyg. and Toxicol.* 31: 352, 1949.

## (79) Ethylenediamine

*ethanediamine, 1,2-diaminoethane**Harmful Effects**Local* Liquid and vapor are irritating to skin, eyes, and mucous membranes. Severe corneal injury and allergic contact dermatitis can occur.*Upper respiratory tract* is irritated by high concentrations.*Route of Entry* Inhalation of vapor.

*Systemic* Headache, vertigo, nausea, and vomiting occur in chronic exposure. Several cases of allergic bronchial asthma have been reported. On the basis of animal experiments, kidney damage may be expected from severe exposures.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

10 parts per million parts of air by volume or 30 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Adhesive workers	Oil neutralizers
Albumin processors	Organic chemical synthesizers
Antifreeze workers	Packagers
Casein processors	Protein processors
Drug makers	Rubber makers
Dye makers	Shellac processors
Emulsion workers	Skin dehairers
Ethylenediamine tetraacetic acid (EDTA) makers	Sulfur processors
Ethylenediamine workers	Surfactant makers
Labelers	Textile lubricant workers

### *Reference*

DERNEHL, C. U.: Clinical experiences with exposures to ethylene amines. *Indust. Med. & Surg.* 20: 541, 1951.

### (80) Ethylene Dibromide

*sym.-dibromoethane, EDB*

### *Harmful Effects*

*Local* Liquid and high vapor concentrations can irritate skin, eyes, and mucous membranes.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Headache, weakness, protracted vomiting, diarrhea, tinnitus, and heart failure. On the basis of animal experiments, irritation of lungs and liver damage may be expected.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

25 parts per million parts of air by volume or 190 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Antiknock compound makers	Lead scavenger makers
Cabbage growers	Motor fuel workers
Celluloid makers	Nematode controllers
Corn growers	Oil processors
Drug makers	Organic chemical synthesizers
Ethylene dibromide workers	Resin makers
Fat processors	Seed corn maggot controllers
Fire extinguisher makers	Soil fumigators
Fruit fumigators	Termite controllers
Fumigant workers	Tetraethyl lead makers
Gasoline blenders	Waterproofing makers
Grain elevator workers	Wax makers
Grain fumigators	Wood insect controllers
Gum processors	Wool reclaimers

*Reference*

OLMSTEAD, E. V.: Pathological changes in ethylene dibromide poisoning. *A.M.A. Arch. Indust. Health* 21: 525, 1960.

**(81) Ethylene Dichloride**

*1,2-dichloroethane, sym.-dichloroethane*

*Harmful Effects*

*Local* Liquid and vapor are irritating to eyes. Irritation by vapor of upper respiratory tract may produce sneezing. Repeated contact with liquid can produce a dry, scaly, fissured dermatitis. Allergic contact dermatitis is rare.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Vapor acts as narcotic in high concentrations and inhalation may produce headache, dizziness, loss of appetite, nausea, vomiting, epigastric pain, visual disturbances, loss of consciousness, and death. Vapor may irritate respiratory tract with production of cough. Liver damage has been suggested by some cases with enlargement of liver and low blood-sugar levels, but ethylene dichloride does not characteristically affect liver. Corneal opacities, as a systemic effect, have been observed only in dogs.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

50 parts per million parts of air by volume or 200 milligrams per cubic meter of air.

## Potential Occupational Exposures

Alkaloid processors	Paint removers
Bakelite processors	Paint remover workers
Camphor workers	Paraffin workers
Cellulose ester workers	Plasticizing bath operators
Cleaning compound makers	Resin makers
Dry cleaners	Rubber makers
Dyers	Soap makers
Ethylene dichloride workers	Solvent workers
Exterminators	Stain removers
Fat processors	Succinic acid makers
Flotation workers	Tetraethyl lead makers
Fumigant workers	Textile cleaners
Gum processors	Tobacco denicotinizers
Insecticide makers	Trichloroethylene makers
Lacquerers	Varnish makers
Lacquer makers	Varnish remover workers
Lacquer remover workers	Vinyl chloride makers
Lead scavenger makers	Wax makers
Metal degreasers	Wire insulators
Oil processors	Wool cleaners
Ore upgraders	

## Reference

IRISH, D. D.: Common chlorinated aliphatic hydrocarbon solvents. *Arch. Environ. Health* 4: 320, 1962.

### (82) Ethylene Glycol

*1,2-ethanediol, glycol alcohol, glycol*

## Harmful Effects

*Local* Liquid may irritate conjunctiva. Skin effects have not been reported.

*Route of Entry* Inhalation of vapor.

*Systemic* Ethylene glycol is a central nervous system depressant producing symptoms similar to ethyl alcohol intoxication. Cases of poisoning have generally followed ingestion of the compound. Inhalation of vapor is uncommon since liquid has high boiling point; however, episodes of unconsciousness, nystagmus, and lymphocytosis have been reported to follow inhalation. Death usually is result of cardiac or renal failure. See Cellosolve.

## Special Diagnostic Test

None.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Antifreeze makers	Lacquerers
Brake fluid makers	Lacquer makers
Cellophane makers	Leather dyers
Cosmetic makers	Metal cleaners
Drug makers	Metal polishers
Dye makers	Painters
Electrolytic condenser makers	Paint makers
Ethylene glycol workers	Resin makers
Explosive makers	Textile makers
Fire extinguisher makers	Tobacco workers
Garage workers	Wax makers
Glue makers	Wood stainers
Glyoxal makers	Wood stain makers
Ink makers	

*References*

MORINI, I.: Several cases of poisoning with commercial ethylene glycol. *Minerva med.* 1: 72, 1954. (*Indust. Hyg. Digest Abst.* No. 210, February 1956)

NADEAU, G.; COTE, R., AND DELANEY, F. J.: Two cases of ethylene glycol poisoning. *Canad. Med. Asso. J.* 70: 69, 1954.

TROISI, F. M.: Chronic intoxication by ethylene glycol vapour. *Brit. J. Indust. Med.* 7: 65, 1950.

**(83) Ethylene Glycol Dinitrate**

*nitroglycol, glycol dinitrate, ethylene dinitrate*

Ethylene glycol dinitrate, itself an explosive, is often used to lower the freezing point of dynamite.

*Harmful Effects*

*Local* None known.

*Routes of Entry* Inhalation of vapor or dust; percutaneous absorption or ingestion of liquid.

*Systemic* EGD is a potent vasodilator and owes much of its toxicity to this property. Acute effects include headache, nausea, vomiting, hypotension, and tachycardia. EGD is a methemoglobin former but methemoglobinemia does not seem to be an important aspect of poisoning. Hypotension is frequently seen in workers exposed daily to EGD. Anginoid pain and cases of sudden death, particularly on hot, humid days, have been reported after removal from daily exposure to EGD following apparent habituation to its vasodilating effects.

### Special Diagnostic Tests

None.

### Threshold Limit Value

(Ethylene glycol dinitrate with nitroglycerine) 0.2 part per million parts of air by volume or 1.2 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### Potential Occupational Exposures

Dynamite makers	Miners
Ethylene glycol dinitrate workers	Powder monkeys

### References

BARSOTTI, M.: Attacks of stenocardia in workers engaged in the production of dynamites with nitroglycol. *Med. Lavoro* 45: 544, 1954.

FRIMMER, M.; GROSS, E.; KIESE, M., AND RESAG, K.: Absorption of ethylene glycol dinitrate through the lung. *Arch. Toxikol.* 18: 200, 1960.

GROSS, E.; BOCK, M., AND HELLRUNG, F.: The toxicology of nitroglycol in comparison with that of nitroglycerin. *Arch. exper. Path. Pharmakol.* 200: 271, 1942.

GROSS, E.; KIESE, M., AND RESAG, K.: Skin absorption of ethylene glycol dinitrate. *Arch. Toxikol.* 18: 194, 1960.

SYMANSKI, H.: Severe injury to health from occupational exposure to nitroglycol. *Arch. Hyg. Bakteriol.* 136: 139, 1952.

VON OETTINGEN, W. F.: The effects of aliphatic nitrous and nitric acid esters on the physiological functions with special reference to their chemical constitution. *Nat. Inst. Health Bull.* No. 186. U.S. Government Printing Office, Washington, D.C., 1946.

## (84) Ethylene Oxide

*1,2-epoxyethane, oxirane, dimethylene oxide*

### Harmful Effects

*Local* Ethylene oxide liquid and gas are irritating to eyes and wet skin, but anhydrous liquid ethylene oxide does not cause primary injury to dry skin. Aqueous solutions near the 50 percent concentration are vesicants. Allergic eczematous dermatitis has also been reported. Ethylene oxide is absorbed by leather and rubber, and may produce belated irritation.

*Route of Entry* Inhalation of gas.

*Systemic* Gas is a pulmonary irritant and in high concentrations will produce pulmonary edema, with cough, dyspnea, and respiratory distress. Systemic effects of headache, nausea, vomiting, and narcosis have been noted. Toxic effects may be due to glycols which are formed when ethylene oxide combines with water in the body.

### Special Diagnostic Test

None.

*Recommended Threshold Limit*

50 parts per million parts of air by volume or 90 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Acrylonitrile makers	Gasoline sweeteners
Butyl cellosolve makers	Grain elevator workers
Detergent makers	Organic chemical synthesizers
Disinfectant makers	Polyglycol makers
Ethanolamine makers	Polyoxirane makers
Ethylene glycol makers	Rocket fuel handlers
Ethylene oxide workers	Rocket fuel makers
Exterminators	Surfactant makers
Farm product fumigators	Textile fumigators
Foodstuff fumigators	Textile lubricant makers
Fumigant makers	Tobacco fumigators
Fungicide workers	

*References*

JACOBSON, K. H.; HACKLEY, E. B., AND FEINSILVER, L.: The toxicity of inhaled ethylene oxide and propylene oxide vapors. *A.M.A. Arch. Indust. Health* 13: 237, 1956.

JACOBSON, K. H.: Industrial hygiene aspects of liquid propellants. *Transactions, 22nd annual meeting, American Conference of Governmental Industrial Hygienists*, 1960. P. 30. Sec.-Treas., 1014 Broadway, Cincinnati 2, Ohio.

ROYCE, A. AND MOORE, W. K. S.: Occupational dermatitis caused by ethylene oxide. *Brit. J. Indust. Med.* 12: 169, 1955.

SEXTON, R. J. AND HENSON, E. V.: Dermatological injuries by ethylene oxide. *J. Indust. Hyg. & Toxicol.* 31: 297, 1949.

## (85) Ethyl Ether

*ethoxyethane, ether, diethyl ether, sulfuric ether, anesthetic ether, ethyl oxide, diethyl oxide*

*Harmful Effects*

*Local* Contact with liquid may produce a dry, scaly, fissured dermatitis.

*Route of Entry* Inhalation of vapor.

*Systemic* In acute exposure, there is a period of excitation followed by central nervous system depression or anesthesia. Pulmonary edema in rare instances may follow acute exposure.

*Special Diagnostic Test*

Analysis of blood for ether. See Von Oettingen, 1958.

*Recommended Threshold Limit*

400 parts per million parts of air by volume or 1,200 milligrams per cubic meter of air.

## Potential Occupational Exposures

Acetic acid makers	Gum processors
Alcohol denaturers	Medical technicians
Alkaloid processors	Motor fuel makers
Anesthetic makers	Nurses
Collodion makers	Oil processors
Drug makers	Organic chemical synthesizers
Dry cleaners	Perfume makers
Ethyl ether workers	Physicians
Explosive makers	Plastic makers
Fat processors	Rayon makers
Fumigant makers	Refrigerant makers
Fumigators	Refrigeration workers
Gasoline engine primers	Wax makers

## References

HAMILTON, A. AND MINOT, G. R.: Ether poisoning in the manufacture of smokeless powder. *J. Indust. Hyg.* 2: 41, 1920.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*, 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(86) Ethyl Mercaptan. *See* Mercaptans

(87) Ethyl Silicate

*tetraethyl orthosilicate, tetraethoxy silane*

## Harmful Effects

*Local* Vapor is irritating to eyes and nose.

*Route of Entry* Inhalation of vapor.

*System* Damage to lungs, liver, and kidney has been observed in experimental animals, but no cases have been reported from industrial exposure.

## Special Diagnostic Test

None.

## Recommended Threshold Limit

100 parts per million parts of air by volume or 850 milligrams per cubic meter of air.

## Potential Occupational Exposures

Acidproof cement makers	Cement preserver makers
Adhesive makers	Ethyl silicate workers
Brick preserver makers	Heat resistant paint makers
Building coaters	Lacquer makers
Casting coaters	Plaster preserver makers

Protective coating makers  
Refractory brick makers  
Silicate paint makers

Stone preserver makers  
Weatherproof cement makers

### Reference

POZZANI, U. C. AND CARPENTER, C. P.: Response of rodents to repeated inhalation of vapors of tetraethyl orthosilicate. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 465, 1951.

## (88) Fluorine and Compounds

### Harmful Effects

*Local* Fluorine gas, anhydrous hydrofluoric acid, and aqueous hydrofluoric acid are intense primary irritants of skin, eyes, and mucous membranes. Burns may be chemical or thermal. Chemical burns cause deep tissue destruction and may not become symptomatic until several hours after contact.

*Route of Entry* Inhalation of gas, mist, dust, or fume.

*Systemic* Fluorine and hydrogen fluoride are pulmonary irritants and produce pulmonary edema. Inhalation of fluoride dust or fume may produce respiratory tract irritation manifested by chills, fever, dyspnea, and cough. Chronic toxicity from inhalation of fluoride as manifested by increased osseous radiopacity is seldom encountered.

### Special Diagnostic Tests

Determination of fluorides in blood and urine; roentgenogram of spine. See Von Oettingen, 1958; Talvitie and Brewer, 1960, and Dubois et al., 1962.

### Recommended Threshold Limit

*Fluorine*, 0.1 part per million parts of air by volume or 0.2 milligram per cubic meter of air.

*Hydrogen fluoride*, 3 parts per million parts of air by volume or 2 milligrams per cubic meter of air.

*Fluoride*, 2.5 milligrams per cubic meter of air.

### Potential Occupational Exposures

#### Fluorine

Antimony fluoride makers  
Cobalt fluoride makers  
Fluoride makers  
Fluorine workers  
Metallic fluoride makers

Rocket fuel handlers  
Rocket fuel makers  
Sulfur hexafluoride makers  
Uranium hexafluoride makers

<i>Hydrogen fluoride</i>	Stainless steel cleaners
Aircraft workers	Stone cleaners
Alloy steel cleaners	Uranium refiners
Aluminum fluoride makers	Yeast makers
Aluminum makers	
Ammonium fluoride makers	
<b>Bleachers</b>	<i>Fluoride</i>
Brass cleaners	Adhesive makers
Brick cleaners	Aluminum anodizers
Casting cleaners	Aluminum makers
Ceramic workers	Aluminum refiners
Copper cleaners	Aluminum solderers
Cryolite makers	Aluminum welders
Crystal glass polishers	Apatite workers
Dye makers	Bactericide workers
Enamel etchers	Beryllium refiners
Fermentation workers	Building workers
Fertilizer makers	Carbon electrode workers
Filter paper makers	Cement workers
Fluoborate makers	Ceramic workers
Fluoride makers	Chemical polisher workers
Fluorine makers	Chlorofluorocarbon makers
Fluorocarbon makers	Concreters
Fluorochemical makers	Construction workers
Fluosilicate makers	Copper refiners
Freon makers	Cryolite makers
Genetron makers	Dentifrice makers
Glass etchers	Diazosalt makers
Graphite purifiers	Disinfectors
Hydrogen fluoride workers	Electric arc welders
Incandescent lamp frosters	Electric arc workers
Isotron makers	Electroplaters
Laundry workers	Electropolishers
Metal cleaners	Embalmers
Metal polishers	Embalming fluid workers
Oil well acidizers	Fluorapatite workers
Ore dissolvers	Fluoride workers
Petroleum refinery workers	Fluorocarbon makers
Plastic makers	Fluorspar miners
Polish workers	Foundry workers
Rocket fuel handlers	Frit workers
Rocket fuel makers	Fungicide workers
	Glass etchers

Gold refiners	Paint workers
Grinding wheel makers	Petroleum refinery workers
Hydrofluoric acid makers	Phosphorescent tube makers
Insect exterminators	Phosphoric acid makers
Insecticide makers	Phosphorus makers
Latex foam rubber workers	Porcelain enamel workers
Laundry workers	Rock phosphate acidulators
Lead electroplaters	Rodent exterminators
Lead smelters	Sandy soil treaters
Light metal casters	Silver refiners
Magnesium foundry workers	Silver solder flux workers
Marble hardeners	Soil improvers
Masonry preservers	Stainless steel welding rod users
Metal cleaners	Super phosphate makers
Metal coating workers	Textile mordanters
Metal refiners	Vegetable growers
Mica makers	Vitreous enamel workers
Mothproofing workers	Water treaters
Opal glass makers	Wood preservative workers
Open hearth steel workers	Wood preservers
Optical equipment makers	Zinc miners

### References

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PRINCI, F.: Fluorides; a critical review. 3, The effects on man of the absorption of fluoride. *J. Occup. Med.* 2: 92, 1960.

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VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. W. B. Saunders Co., Philadelphia, 1958.

### (89) Formaldehyde

*methanal, oxomethane, oxymethylene, methylene oxide, formic aldehyde, methyl aldehyde*

Formalin is a 40 percent aqueous solution of formaldehyde.

### Harmful Effects

*Local* Formaldehyde gas is highly irritating to conjunctiva and mucous membranes of upper respiratory tract. Aqueous solutions may cause

contact dermatitis from primary irritation or allergic hypersensitivity. Urticaria has been reported following inhalation of gas.

*Route of Entry* Inhalation of gas.

*Systemic* Systemic intoxication appears to be of little importance in industry since intense irritation of upper respiratory tract compels worker to leave the area. Ingestion may result in gastrointestinal irritation, respiratory depression, and death.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

5 parts per million parts of air by volume or 6 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Anatomists	Furniture sprayers
Bakers	Fur processors
Biologists	Glass etchers
Bookbinders	Hexamethylenetetramine makers
Botanists	Hide preservers
Crease-resistant textile finishers	Histology technicians
Deodorant makers	Ink makers
Disinfectant makers	Lacquerers
Disinfectors	Lacquer makers
Dress goods store personnel	Oil well workers
Dress makers	Paper makers
Drug makers	Pentaerythritol makers
Dye makers	Photographic film makers
Embalmers	Resin makers
Embalming fluid makers	Rubber makers
Ethylene glycol makers	Tannery workers
Formaldehyde resin makers	Textile mordanters
Formaldehyde workers	Textile printers
Fungicide workers	Textile waterproofers
Furniture dippers	Wood preservers

### *References*

GLASS, W. I.: An outbreak of formaldehyde dermatitis. *New Zealand J. Med.* 60: 423, 1961.

HENSON, E. V.: The toxicology of some aliphatic aldehydes. *J. Occup. Med.* 1: 457, 1959.

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ROY, M., JR.: Corrosive gastritis after formaldehyde ingestion; report of a case. *New Eng. J. Med.* 266: 1248, 1962.

## (90) Formic Acid

*methanoic acid, formylic acid, hydrogen carboxylic acid**Harmful Effects*

*Local* Formic acid vapor is irritating to mucous membranes of upper respiratory tract. Liquid in concentrated solution is primary skin irritant.

*Routes of Entry* Percutaneous absorption of liquid; inhalation of vapor. *Systemic* Systemic effects have not been recognized in industry. Fatal poisoning from accidental or suicidal ingestion of formic acid has been frequently encountered. The clinical picture is characterized by salivation, burning sensation in mouth and pharynx, vomiting, hematemesis, diarrhea, and severe abdominal pain. Shock may result with subsequent acute renal failure, or respiratory failure and death.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Acetic acid makers	Leather makers
Airplane dope makers	Nickel platers
Allyl alcohol makers	Ore refiners
Cellulose formate makers	Organic ester makers
Dyers	Oxalic acid makers
Electroplaters	Paper makers
Food preservers	Perfume makers
Formate makers	Phenolic resin makers
Formic acid workers	Refrigerant makers
Fumigant makers	Rubber workers
Glass silverers	Tannery workers
Insecticide makers	Textile makers
Lacquer makers	Wine makers
Laundry workers	

*Reference*

HENSON, E. V.: Toxicology of the fatty acids. *J. Occup. Med.* 1: 339, 1959.

(91) Freon<sup>R</sup>

*Freon-11*, fluorotrichloromethane

*Freon-12*, dichlorodifluoromethane

*Freon-12B2*, difluorodibromomethane

*Freon-13*, monochlorotrifluoromethane

*Freon-13B1*, trifluoromonobromomethane

*Freon-14*, tetrafluoromethane

*Freon-21*, dichloromonofluoromethane

*Freon-22*, monochlorodifluoromethane

*Freon-23*, trifluoromethane

*Freon-112*, tetrachlorodifluoroethane

*Freon-113*, trichlorotrifluoroethane

*Freon-113B2*, dibromomonochlorotrifluoroethane

*Freon-114*, dichlorotetrafluoroethane

*Freon-114B2*, dibromotetrafluoroethane

*Freon-115*, monochloropentafluoroethane

*Freon-C318*, octafluorocyclobutane

### *Harmful Effects*

*Local* These fluorinated hydrocarbons may produce very mild irritation of the upper respiratory tract. If chlorine-containing fluoromethanes come into contact with an open flame or hot metal, the decomposition products of hydrogen chloride, hydrogen fluoride, phosgene, sulfur dioxide and chlorine may cause severe irritative effects.

*Route of Entry* Inhalation of vapor or gas.

*Systemic* Certain of these Freons may produce mild central nervous system depression. Systemic effect may be due in part to displacement of air, with resultant hypoxia.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

*Freon-11*, 1,000 parts per million parts of air by volume or 5,600 milligrams per cubic meter of air.

*Freon-12*, 1,000 parts per million parts of air by volume or 4,950 milligrams per cubic meter of air.

*Freon-12B2*, 100 parts per million parts of air by volume or 860 milligrams per cubic meter of air.

*Freon-13B1*, 1,000 parts per million parts of air by volume or 6,100 milligrams per cubic meter of air.

*Freon-21*, 1,000 parts per million parts of air by volume or 4,200 milligrams per cubic meter of air.

*Freon-112* (tentative), 500 parts per million parts of air by volume or 4,170 milligrams per cubic meter of air.

*Freon-113*, 1,000 parts per million parts of air by volume or 7,600 milligrams per cubic meter of air.

*Freon-114*, 1,000 parts per million parts of air by volume or 7,000 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Aerosol bomb workers	Plastic makers
Ceramic mold makers	Pressurized food makers
Drug makers	Refrigerant workers
Fire extinguisher workers	Rocket fuel makers
Freon workers	Solvent workers
Heat transfer workers	Sponge rubber makers
Metal conditioners	

*References*

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PATTISON, F. L. M.: *Toxic Aliphatic Fluorine Compounds*. Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1959.

## (92) Furfural

*furfurol, furfuraldehyde, artificial ant oil, pyromucic aldehyde, furol*

*Harmful Effects*

*Local* Liquid and high vapor concentrations are irritating to skin, eyes, and mucous membranes; can produce corneal anesthesia. Allergic contact dermatitis and photosensitization may occur.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Acute effects from inhalation of vapor have been limited to headaches and breathing difficulties.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

5 parts per million parts of air by volume or 20 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Adipic acid makers	Lubricating oil refiners
Adiponitrile makers	Lysine makers
Butadiene refiners	Metal refiners
Cellulose acetate makers	Nitrocellulose makers
Disinfectant workers	Nylon makers
Disinfectors	Paint remover makers
Fungicide workers	Petroleum refinery workers
Furfural workers	Phenol furfural makers
Grinding wheel makers	Rare earth refiners
Herbicide makers	Resin makers
Highway maintenance workers	Road builders

Rosin refiners  
Rubber makers  
Shoe dye makers  
Varnish makers

Weed sprayers  
Wetting agent workers  
Wood rosin decolorizers

## Reference

DUNLOP, A. P. AND PETERS, F. N.: *The Furanes*. American Chemical Society Monograph Series No. 119. Reinhold Publishing Corp., New York, 1953.

## (93) Gasoline

*petrol, motor spirits*

### Harmful Effects

*Local* Gasoline is irritating to skin, conjunctiva, and mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of vapor. Ill effects from percutaneous absorption of liquid are questionable.

*Systemic* Exposure to low concentrations of vapor may produce symptoms similar to ethyl alcohol intoxication, including flushing of face, staggering gait, slurred speech, and mental confusion. Higher concentrations may result in unconsciousness, coma, and death. Ingestion of liquid often results in aspiration with a pneumonitis similar to that seen in kerosine intoxication. Symptoms of gastrointestinal irritation may also occur.

The existence of chronic poisoning has been questioned. The possibility of blood alterations developing from absorption of aromatic hydrocarbons in gasoline should be considered.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

500 parts per million parts of air by volume or 2,000 milligrams per cubic meter of air.

### Potential Occupational Exposures

Gasoline is used as a fuel, diluent, and solvent in numerous occupations throughout various industries.

### References

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MACHEL, W.: Gasoline intoxication. *J. Am. Med. Assoc.* 117: 1065, 1941.

TUCKER, R.; KILBOURNE, E. D., AND EVANS, J. B.: Pulmonary manifestations of gasoline intoxication. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 2: 17, 1950.

WANG, C. C. AND IRONS, G. V.: Acute gasoline intoxication. *Arch. Environ. Health* 2: 714, 1961.

## (94) Germanium Compounds

*Harmful Effects*

*Local* Germanium tetrachloride and tetrafluoride are mucous membrane irritants. No local effects of other germanium compounds have been reported.

*Route of Entry* Inhalation of gas or vapor.

*Systemic* No cases of industrial poisoning have been reported.

*Special Diagnostic Tests*

Analysis of urine and feces for germanium. *See* Fairhall, 1957.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Argyrodite workers	Photodiode makers
Dental alloy makers	Rectifier makers
Feldistor makers	Semiconductor makers
Germanite workers	Transistor makers
Germanium workers	Vacuum tube makers
Glass makers	Zinc residue workers
Phosphor makers	

*References*

DUDLEY, H. C. AND WALLACE, E. J.: Pharmacological studies of radio-germanium (Ge-71). *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 263, 1952.

FAIRHALL, L. T.: *Industrial Toxicology*. 2nd ed. Williams & Wilkins Co., Baltimore, 1957.

HARROLD, G. C. AND MEEK, S. F.: The physiologic properties of germanium. *Indust. Med.* 13: 236, 1944.

HUEPER, W. C.: Germanium. *Occup. Med.* 4: 208, 1947.

## (95) Graphite

*plumbago, black lead, mineral carbon*

*Harmful Effects*

*Local* None.

*Route of Entry* Inhalation of dust.

*Systemic* Natural graphite contains free crystalline silica and exposures of several years to this silica-graphite combination may produce a disabling pneumoconiosis that is similar to other modified silica pulmonary reactions, for example, anthracosilicosis. Pure graphite has not been shown to produce this picture of severe pulmonary change.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Battery makers, dry	Match makers
Brake lining makers	Nuclear reactor workers
Cathode ray tube makers	Paint makers
Commutator brush makers	Pencil lead makers
Crucible makers	Pigment makers
Electric appliance makers	Pipe joint compound makers
Electrode makers	Polish makers
Electroplaters	Radio resistor makers
Electrotypes	Refractory material makers
Explosive makers	Retort makers
Foundry workers	Roofing makers
Gasket makers	Steel makers
Graphite cement makers	Stove polish makers
Graphite miners	Thermocouple (with tungsten) makers
Graphite workers	
Lubricant makers	

*References*

HARDING, H. E. AND OLIVER, G. B.: Changes in the lungs produced by natural graphite. *Brit. J. Indust. Med.* 6: 91, 1949.

HIRSCH, M. J.; KASS, I.; SCHAEFER, W. B., AND DENST, J.: Infection with atypical tubercle bacilli in graphite pneumoconiosis. *A.M.A. Arch. Int. Med.* 103: 814, 1959.

**(96) Hexamethylenetetramine**

*methenamine, hexamine, formamine, ammonioformaldehyde*

*Harmful Effects*

*Local* Contact with solid, or its fumes when heated, can irritate skin or produce an allergic eczematous contact dermatitis.

*Route of Entry* Ingestion.

*Systemic* Gastrointestinal irritation and kidney damage have occurred following large oral doses.

*Special Diagnostic Tests*

None.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Anticorrosion additive workers	Phenol-formaldehyde resin workers
Drug makers	Phosgene absorption cannister makers
Explosive makers	Resin makers
Foundry workers	Rubber makers
Fuel tablet makers	Textile makers
Fungicide makers	Urea-formaldehyde resin workers
Gas mask makers	Veterinarians
Hexamethylenetetramine workers	

*Reference*

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(97) *Hydrazine**hydrazine base, diamine**Harmful Effects*

*Local* Contact of this hygroscopic liquid with skin and eyes produces penetrating burns. Contact with vapor results in eczematous dermatitis from either primary irritation or allergic hypersensitivity. Irritation of eyes and nose by high concentrations is so intense as to compel workers to leave the area usually before lower respiratory tract suffers damage.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Low grade exposure produces headache, nausea, and dizziness. Bronchitis and pneumonitis may result if early irritative warnings are not heeded. On basis of animal experiments, hydrazine may also produce central nervous system symptoms of excitement and convulsions, fatty necrosis of liver, nephritis, hemolytic anemia, hypoglycemia, and hypotension.

*Special Diagnostic Test*

Pyridyl test for hydrazine assay in blood plasma. See Prescott et al., 1955.

*Recommended Threshold Limit*

1 part per million parts of air or 1.3 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Agricultural chemical makers	Drug makers
Anticorrosion additive workers	Explosive makers
Antioxidant workers	Hydraulic fluid workers
Boiler operators	Hydrazine workers
Chlorine scavenger makers	Insecticide makers

Jet fuel handlers	Solder flux makers
Jet fuel makers	Sponge rubber makers
Oxygen scavenger makers	Textile dyers, acrylic and vinyl
Photographic developer makers	Vat dye makers
Rocket fuel handlers	Water treaters
Rocket fuel makers	

## References

EVANS, D. M.: Two cases of hydrazine hydrate dermatitis without systemic intoxication. *Brit. J. Indust. Med.* 16: 126, 1959.

JACOBSON, K. H.: Industrial hygiene aspects of liquid propellants. In *Transactions, 22nd annual meeting, American Conference of Governmental Industrial Hygienists, 1960*. Sec.-Treas., 1014 Broadway, Cincinnati 2, Ohio.

KROP, S.: Toxicity of hydrazine. A review. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 9: 199, 1954.

OFFICE OF DIRECTOR, DEFENSE RESEARCH AND ENGINEERING, DEPARTMENT OF DEFENSE: *The Handling and Storage of Liquid Propellants*. U.S. Government Printing Office, Washington, D.C., 1961.

PREScott, B.; KAUFFMANN, G., AND JAMES, W. D.: The "pyridyl" test for hydrazine assay in blood plasma. *A.M.A. Arch. Indust. Health* 12: 393, 1955.

SCHULTHEISS, E.: Hypersensitivity to hydrazine. *Berufsdermatosen* 7: 131, 1959.

(98) Hydrogen Bromide. *See* Bromine and Compounds

(99) Hydrogen Chloride

*anhydrous hydrochloric acid, chlorohydric acid*

An aqueous solution of hydrogen chloride gas is hydrochloric acid or muriatic acid.

### Harmful Effects

*Local* Hydrochloric acid and high concentrations of hydrogen chloride gas are highly irritating to eyes, skin, and mucous membranes. Discoloration of teeth and tooth decay have been noted from exposure to low concentrations of gas.

*Route of Entry* Inhalation of gas or mist.

*Systemic* Pulmonary edema is possible, but usually the cough and choking sensation from intense irritation of upper respiratory tract compel worker to leave the area.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

(Hydrogen chloride) 5 parts per million parts of air by volume or 7 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Alkyl chloride makers	Lithographers
Battery makers	Metal cleaners
Bleachers	Oil well treaters
Boiler scale removers	Ore reduction workers
Bronzers	Organic chemical synthesizers
Chloride makers	Photoengravers
Chloroprene makers	Pigment workers
Corn syrup makers	Plastic workers
Drug makers	Pottery workers
Dye makers	Rubber makers
Electroplaters	Silica gel makers
Enamelters	Soap makers
Fertilizer makers	Sugar cane refiners
Food processors	Tannery workers
Galvanizers	Tantalum ore refiners
Gas well treaters	Tetraethyl lead makers
Glass finishers	Textile workers
Glass mixers	Tin ore refiners
Glue makers	Veterinarians
Hydrogen chloride workers	Vinyl chloride makers
Jewelers	Wire annealers

*References*

QUERIES AND MINOR NOTES: Effects of hydrochloric acid fumes. *J. Am. Med. Assoc.* 131: 1182, 1946.

THIELE, E.: Fatal poisoning from use of hydrochloric acid in a confined space. *Zentralbl. Arbeitsmed. u. Arbeitsschutz* 3: 146, 1953. ( *Indust. Hyg. Digest*, Abst. No. 387, April 1954)

(100) Hydrogen Cyanide  
*prussic acid, hydrocyanic acid*

*Harmful Effects*

*Local* None.

*Route of Entry* Inhalation of gas and percutaneous absorption of gas or liquid.

*Systemic* Symptoms are caused by chemical asphyxia, that is, inhibition of cellular oxidative processes. Acute and subacute symptoms include headache, lassitude, nausea, vomiting, shortness of breath, irritation of throat, convulsions, respiratory paralysis, coma, and death. Chronic toxicity is debatable. In general, systemic toxicity is similar for other cyanides and cyanogen compounds.

*Special Diagnostic Tests*

Cyanide determination in blood and tissues; thiocyanate determination in serum and urine. See Amdur, 1959, and Elkins, 1959.

*Recommended Threshold Limit*

10 parts per million parts of air by volume or 11 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Acid dippers	Heat treaters
Acrylate makers	Hexamethylenediamine makers
Acrylonitrile makers	Hydrocyanic acid makers
Adipic acid makers	Hydrogen cyanide workers
Adiponitrile makers	Insecticide makers
Aircraft workers	Jewelers
Ammonium salt makers	Metal cleaners
Art printing workers	Metal polishers
Blacksmiths	Methacrylate makers
Blast furnace workers	Mirror silverers
Bone distillers	Mordanters
Bronzers	Nylon makers
Browners, gun barrel	Organic chemical synthesizers
Cadmium platers	Oxalic acid makers
Case hardeners	Phosphoric acid makers
Cellulose product treaters	Photoengravers
Coal tar distillery workers	Pigment makers
Coke oven operators	Plastic workers
Cyanide workers	Polish makers
Cyanogen makers	Rayon makers
Disinfectant makers	Rubber makers
Dye makers	Silver extractors
Electroplaters	Silver refiners
Exterminators	Solderers
Fertilizer makers	Steel carburizers
Fulminate mixers	Tannery workers
Fumigant makers	Temperers
Fumigators	Textile printers
Gas purifiers	Tree sprayers
Gas workers, illuminating	White cyanide makers
Gilders	Zinc platers
Gold extractors	Zinkers
Gold refiners	

*References*

AMDUR, M. L.: Accidental exposure to acetonitrile; a clinical study. *J. Occup. Med.* 1: 627, 1959.

ELKINS, H. B.: *The Chemistry of Industrial Toxicology*. 2nd ed. John Wiley and Sons, New York, 1959.

WOLFSIE, J. H. AND SHAFFER, C. B.: Hydrogen cyanide; hazards, toxicology, prevention and management of poisoning. *J. Occup. Med.* 1: 281, 1959.

(101) Hydrogen Fluoride. *See* Fluorine and Compounds

(102) Hydrogen Peroxide

*peroxide, hydrogen dioxide*

### *Harmful Effects*

*Local* Concentrated liquid and mist are extremely caustic to skin and eyes. Damage to eyes may be delayed in appearance.

*Route of Entry* Inhalation of vapor or mist.

*Systemic* Inhalation of vapor or mist produces effects ranging from mild bronchitis to pulmonary edema. No chronic systemic effects have been observed.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

(Hydrogen peroxide, 90 percent) 1 part per million parts of air by volume or 1.4 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Acetone makers	Hide disinfectors
Alcoholic liquor agers	Hydrogen peroxide workers
Antichlor makers	Ivory bleachers
Antiseptic makers	Metal cleaners
Benzol peroxide makers	Oil painting renovators
Bleachers	Oil refiners
Bone bleachers	Photographic film developers
Button makers	Plastic foam makers
Disinfectant makers	Rocket fuel handlers
Drug makers	Rocket fuel makers
Dyers	Silk bleachers
Electroplaters	Soap bleachers
Fat refiners	Sponge rubber makers
Feather bleachers	Straw bleachers
Felt hat makers	Textile bleachers
Flour bleachers	Torpedo propellant workers
Fruit bleachers	Veterinarians
Fruit preservers	Water treaters
Fur bleachers	Wax bleachers
Fur dyers	Wine agers
Gelatin bleachers	Wood pulp bleachers
Glue bleachers	Wool printers
Hair bleachers	

## (103) Hydrogen Sulfide

*sulfuretted hydrogen, stink damp*

Hydrogen sulfide is usually encountered as an industrial byproduct, but also occurs in mines, natural gas, and crude oil, and is formed from decomposing sewage and other organic matter. Certain chemicals such as thioglycolic acid and lithopone liberate hydrogen sulfide on decomposition.

*Harmful Effects*

*Local* Irritating to eyes and to mucous membranes of nose and throat.

*Route of Entry* Inhalation of gas.

*Systemic* Hydrogen sulfide is an asphyxiant because of its ability to paralyze the respiratory centers of brain with resultant cessation of respiration. Unless death occurs during period of respiratory paralysis, recovery is usually complete. An exception to this tendency toward complete recovery is occasionally seen when period of hypoxia produces permanent brain injury.

Prolonged exposure to moderately high concentrations of hydrogen sulfide may irritate tissues of respiratory tract sufficiently to produce pneumonitis or pulmonary edema. Excessive exposure to concentrations of this order of magnitude may also be attended by such symptoms as headache, gastrointestinal disturbances, dizziness, chest pain, and cough.

Although the physiologic response to different concentrations of hydrogen sulfide is subject to considerable individual variation, the accompanying table indicates the general response that might be expected to occur at a given concentration.

Parts per million	Percent	Response
0.20	0.00002	Detectable odor
20	0.002	Maximum allowable concentration for daily 8-hour exposure
150	0.015	Olfactory nerve paralysis
250	0.025	Prolonged exposure may cause pulmonary edema
500	0.05	Systemic symptoms may occur in $\frac{1}{2}$ to 1 hour
1,000	0.10	Rapid collapse; respiratory paralysis imminent
5,000	0.5	Immediate death

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

20 parts per million parts of air by volume or 30 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Barium carbonate makers	Manholes, workers in
Barium salt makers	Miners
Blast furnace workers	Natural gas makers
Brewery workers	Paper pulp makers
Cable splicers	Petroleum refinery workers
Caisson workers	Phosphate purifiers
Carbon disulfide makers	Photoengravers
Cellophane makers	Pyrite burners
Cistern cleaners	Rayon makers
Coke oven workers	Refrigerant makers
Copper ore sulfidizers	Septic tank cleaners
Depilatory makers	Sewage treatment plant workers
Dye makers	Sewer workers
Fat renderers	Sheep dippers
Felt makers	Silk makers
Fertilizer makers	Slaughterhouse workers
Fur dressers	Soap makers
Glue makers	Sugar beet processors
Gold ore workers	Sulfuric acid purifiers
Heavy metal precipitators	Sulfur makers
Hydrochloric acid purifiers	Synthetic fiber makers
Hydrogen sulfide workers	Tannery workers
Laboratory workers, chemical	Textile printers
Lead ore sulfidizers	Tunnel workers
Lead removers	Vulcanizers
Lithographers	Well diggers
Lithopone makers	

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Mexico, An evaluation of the incident of Nov. 24, 1950. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 199, 1952.

MILBY, T. H.: Hydrogen sulfide intoxication; review of the literature and report of unusual accident resulting in two cases of nonfatal poisoning. *J. Occup. Med.* 4: 431, 1962.

YANT, W. P.: Hydrogen sulfide in industry; occurrence, effects, and treatment. *Am. J. Pub. Health* 20: 598, 1930.

### (104) Hydroquinone

*quinol, hydroquinol, paradiphenol, hydrochinone, dihydroxybenzene*

#### *Harmful Effects*

*Local* Contact dermatitis due to either primary irritation or allergic hypersensitivity. Eye irritation manifested by conjunctivitis, lacrimation, photophobia, corneal stains, and opacities. Reddish discoloration of hair and exposed skin. Skin may be depigmented by contact with hydroquinone as well as with the monobenzyl ether derivative.

*Route of Entry* Inhalation of dust.

*Systemic* Based on symptoms following ingestion, inhalation of hydroquinone may produce blurred speech, tinnitus, dyspnea, tremors, convulsions, cyanosis from methemoglobinemia, and hemolytic anemia.

#### *Special Diagnostic Test*

Detection of hydroquinone in urine. See Von Oettingen, 1958.

#### *Recommended Threshold Limit*

2 milligrams per cubic meter of air.

#### *Potential Occupational Exposures*

Ceramic decorators	Photographic developer makers
Drug makers	Plastic makers
Dye makers	Plastic stabilizer workers
Fatty oil processors	Rubber coating workers
Fur dyers	Stone coating workers
Hydroquinone workers	Styrene monomer workers
Lubricating oil workers	Textile coating workers
Motor fuel blenders	Varnish makers
Paint makers	

#### *References*

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VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (105) Iron Compounds

*Harmful Effects*

*Local* Ferric chloride, ferric ferrocyanide, and ferric sesquichloride are known skin sensitizers.

*Route of Entry* Inhalation of dust.

*Systemic* Iron salts may irritate respiratory tract. Iron oxide, when inhaled, may produce roentgenographic changes in lungs which resemble silicosis. This condition is referred to as siderosis and is thought to be benign. See Pulmonary Siderosis, Pneumoconioses section. Iron carbonyl is a liquid with highly toxic vapors which, upon inhalation, may produce extreme pulmonary irritation.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

(Iron oxide fume) 15 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Arc cutters	Friction saw operators
Arc welders	Iron workers
Bessemer operators	Metalizers
Buttwelders	Oxyacetylene cutters
Electric arc welders	Seam welders
Electric furnace operators	Stainless steel makers
Flame cutters	Steel foundry workers

## (106) Isopropyl Acetate

*Harmful Effects*

*Local* Vapor can be irritating to conjunctiva and to mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of vapor.

*Systemic* No ill effects from use of isopropyl acetate in industry have been recorded. Vapors can produce central nervous system depression following excessive exposure.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

Not established.

## Potential Occupational Exposures

Dope processors	Oil processors
Fat processors	Organic chemical synthesizers
Gum processors	Perfume makers
Isopropyl acetate workers	Plastic makers
Lacquerers	Resin makers
Lacquer makers	Silk makers
Leather makers, artificial	Solvent workers
Nitrocellulose makers	Wax makers

### (107) Isopropyl Alcohol

*isopropanol, 2-propanol, secondary propyl alcohol, dimethyl-carbinol*

#### Harmful Effects

*Local* Inhalation of vapor can produce mild irritation of conjunctiva and mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of vapor.

*Systemic* No industrial poisoning has been recorded. Isopropyl alcohol is potentially narcotic.

#### Special Diagnostic Tests

Analysis of isopropyl alcohol and acetone in blood, urine and body tissues.

See Patty, 1949.

#### Recommended Threshold Limit

400 parts per million parts of air by volume or 980 milligrams per cubic meter of air.

## Potential Occupational Exposures

Acetone makers	Lacquer makers
Alkaloid processors	Nurses
Antifreeze makers	Oil processors
Cosmetic makers	Perfume makers
De-icing compound makers	Photographic film developers
Drug makers	Physicians
Gasoline makers	Resin makers
Glass makers	Rocket fuel handlers
Gum processors	Rocket fuel makers
Ink makers	Solvent workers
Isopropyl alcohol workers	Stainers
Laboratory workers, chemical	Stain makers
Lacquerers	Varnish makers

## References

HENSON, E. V.: The toxicology of some aliphatic alcohols; part 2. *J. Occup. Med.* 2: 497, 1960.

PATTY, F. A. (EDITOR): *Industrial Hygiene and Toxicology*. 1st ed., vol. 2. Interscience Publishers, New York, 1949.

### (108) Kerosine (kerosene)

#### Harmful Effects

*Local* Contact with liquid may produce primary skin irritation.

*Route of Entry* Inhalation of vapor; ingestion of liquid.

*Systemic* Toxic manifestations include central nervous system depression and pneumonia. Pulmonary effects may follow aspiration of liquid accidentally ingested.

#### Special Diagnostic Test

None.

#### Recommended Threshold Limit

Not established.

#### Potential Occupational Exposures

Farmers	Kerosine workers
Garage workers	Metal cleaners
Heating fuel handlers	Petroleum refinery workers
Insecticide workers	Rocket fuel handlers
Jet fuel handlers	Rocket fuel makers
Jet fuel makers	

#### Reference

HENSON, E. V.: Toxicology of some of the aliphatic and alicyclic hydrocarbons. *J. Occup. Med.* 1: 105, 1959.

### (109) Ketones

Commonly used ketone solvents include

*acetone* (dimethyl ketone, beta-ketopropane, pyroacetic ether)

*butanone* (methyl ethyl ketone, MEK, ethyl methyl ketone)

*pentanone* (methyl propyl ketone, MPK, ethyl acetone)

*methyl butyl ketone* (propyl acetone)

#### Harmful Effects

*Local* These solvents can produce a dry, scaly, and fissured dermatitis after repeated exposure. High vapor concentrations may irritate conjunctive and mucous membranes of nose and throat.

*Route of Entry* Inhalation of vapor.

*Systemic* In high concentrations, narcosis is produced, with symptoms of headache, nausea, vomiting, dizziness, incoordination, and unconsciousness.

### *Special Diagnostic Tests*

Acetone, determination of acetone in blood and urine. *See Von Oettingen, 1958.*

### *Recommended Threshold Limits*

*Acetone*, 1,000 parts per million parts of air by volume or 2,400 milligrams per cubic meter of air.

*Butanone*, 200 parts per million parts of air by volume or 590 milligrams per cubic meter of air.

*Pentanone*, 200 parts per million parts of air by volume or 700 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

#### *Acetone*

Acetic acid makers	Mesityl oxide makers
Acetic anhydride makers	Metal cleaners
Acetone workers	Methyl isobutyl ketone makers
Acetylene cylinder fillers	Methyl methacrylate workers
Adhesive makers	Painters
Bronzers	Paint makers
Celluloid makers	Paint remover workers
Cellulose acetate makers	Paraffin processors
Chloroform makers	Pesticide makers
Diacetone alcohol makers	Photographic film makers
Drug makers	Phorone makers
Electronic equipment cleaners	Resin makers
Electronic equipment dryers	Rubber cement workers
Explosive makers	Rubber workers
Glycol makers	Solvent workers
Iodoform makers	Stainers
Isoprene makers	Stain makers
Lacquerers	Textile makers
Lacquer makers	Varnish makers
Lubricating oil dewaxers	Varnish remover workers

#### *Butanone*

Adhesive makers	Cosmetic makers
Butanone workers	Dewaxers
Cellulose cement makers	Dope processors
Cleaning compound makers	Drug makers
Colorless synthetic resin makers	Dye makers

Explosive makers	Printing ink makers
Lacquerers	Raincoat makers
Lacquer makers	Rubber makers
Lacquer remover workers	Shoemakers
Leather workers, artificial	Smokeless powder makers
Oil processors	Solvent workers
Organic chemical synthesizers	Stainers
Painters	Stain makers
Paint remover makers	Varnish makers
Petroleum refinery workers	Varnish remover workers
Photographic film makers	Vinyl raincoat makers
Printers	
<i>Pentanone</i>	
Pentanone workers	Solvent workers

### References

HENSON, E. V.: Toxicology of some aliphatic ketones. *J. Occup. Med.* 1: 607, 1959.  
 VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (110) Lead

### Harmful Effects

*Local* None.

*Routes of Entry* Ingestion of dust; inhalation of dust or fume.

*Systemic* Lead poisoning in industry almost always results from inhalation of lead-containing dust or lead fume. Signs and symptoms of lead poisoning may include abdominal pain (colic) with tenderness, constipation, headache, weakness, muscular aches or cramps, loss of appetite, nausea, vomiting, weight loss, anemia with pallor, and a *lead line* of the gingival margin. Lead palsy and lead encephalopathy resulting from industrial exposure occur infrequently. See Tetraethyl Lead, and Tetramethyl Lead.

### Special Diagnostic Tests

Analysis of blood and urine for lead, and of urine for coproporphyrins; examination of blood smear for stipple cells. See Kehoe, 1951.

### Recommended Threshold Limit

0.2 milligram per cubic meter of air.

### Potential Occupational Exposures

Babbitters	Brass founders
Battery makers	Brass polishers
Bookbinders	Braziers
Bottle cap makers	Brick burners

Brick makers	Lead mill workers
Bronzers	Lead miners
Brush makers	Lead pipe makers
Cable makers	Lead salt makers
Cable splicers	Lead shield makers
Canners	Lead smelters
Cartridge makers	Lead stearate makers
Ceramic makers	Lead workers
Chemical equipment makers	Linoleum makers
Chippers	Linotypers
Cutlery makers	Lithographers
Demolition workers	Match makers
Dental technicians	Metal burners
Diamond polishers	Metal cutters
Dye makers	Metal grinders
Electronic device makers	Metal miners
Electroplaters	Metal polishers
Electrotypes	Metal refiners
Emery wheel makers	Mirror silverers
Enamel burners	Motor fuel blenders
Enamelers	Musical instrument makers
Enamel makers	Painters
Farmers	Paint makers
File cutters	Paint pigment makers
Filers	Patent leather makers
Flower makers, artificial	Pearl makers, imitation
Foundry molders	Pipe fitters
Galvanizers	Plastic workers
Glass makers	Plumbers
Glass polishers	Pottery glaze mixers
Gold refiners	Pottery workers
Gun barrel browners	Putty makers
Incandescent lamp makers	Riveters
Insecticide makers	Roofers
Insecticide users	Rubber buffers
Japan makers	Rubber makers
Japanners	Scrap metal workers
Jewelers	Sheet metal workers
Junk metal refiners	Shellac makers
Lacquer makers	Ship dismantlers
Lead burners	Shoe stainers
Lead counterweight makers	Shot makers
Lead flooring makers	Solderers
Lead foil makers	Solder makers

Steel engravers	Tinners
Stereotypers	Type founders
Tannery workers	Typesetters
Temperers	Varnish makers
Tetraethyl lead makers	Wallpaper printers
Tetramethyl lead makers	Welders
Textile makers	Zinc mill workers
Tile makers	Zinc smelter chargers
Tin foil makers	

### References

KEHOE, R. A.: A critical appraisal of current practices in the clinical diagnosis of lead intoxication. *Indust. Med. & Surg.* 20: 253, 1951.

KEHOE, R. A.: Lead poisoning. In Cecil, R. L. and Loeb, R. F. (editors): *Textbook of Medicine*. 10th ed. W. B. Saunders Co., Philadelphia, 1959.

SASSI, C.; FINULLI, M., AND NAVA, C.: Saturnism in the processing of lead stearate. *Med. Lavoro* 52: 658, 1961.

SKINNER, H. L., JR.: The lead problem. An outline of current knowledge and opinion. *J. Occup. Med.* 3: 429, 1961.

VARIOUS AUTHORS: *Lead Symposium*, February 25-27, 1963. University of Cincinnati, Cincinnati, Ohio, 1963.

ZIMMER, F. E.: Lead poisoning in scrap-metal workers. *J. Am. Med. Assoc.* 175: 238, 1961.

(111) Lindane. *See* Pesticides Section

(112) Manganese Compounds

### Harmful Effects

*Local* Manganese dust may be irritating to upper respiratory tract.

*Route of Entry* Inhalation of dust or fume.

*Systemic* Among the various manganese compounds used in industry, the oxides of manganese have been almost exclusively responsible for the development of disease. Symptoms generally appear between one and two years following initial exposure. Workers appear to vary in their susceptibility to these compounds.

The course of chronic manganese intoxication may be divided into three phases.

(1) Prodromal phase—characterized by insidious onset and subjective symptoms of headache, asthenia, anorexia, apathy, insomnia or somnolence, leg cramps, impotence, and a diminished desire to talk.

(2) Intermediate phase—characterized by objective symptoms and signs of speech disturbances (monotonous speech, slowness, poor articulation, stuttering, blocking, muteness), masklike face, spasmodic laughing, euphoria, slow and clumsy movements, diminished reflexes, and gait disturbances.

(3) Established phase—symptoms and signs may be exaggerated. Gait disturbances may consist of slow, spasmotic, staggering, high-stepping, or swinging gait. Falls are frequent. Tremors of extremities may appear. Central nervous system manifestations are often permanent resulting in partial or total disability.

There is no unanimity of opinion on the relationship between manganese and pneumonia. A report on manganese pneumonitis, published in 1946, disclosed that men exposed to inhalation of oxide dust suffered a pneumonia rate that averaged 26 per thousand workers over the seven years 1938-1945 as compared with 0.73 per thousand in a control group. No permanent pulmonary changes were observed in exposed group, either on clinical or radiologic examination.

### Special Diagnostic Tests

Analysis of blood, urine, and feces for manganese. *See Bolton et al., 1962.*

### Recommended Threshold Limit

(Manganese) 5 milligrams per cubic meter of air.

### Potential Occupational Exposures

Battery makers	Manganese ore miners
Brick makers	Manganese ore smelters
Ceramic makers	Manganese soap makers
Copper manganese alloy makers	Manganese steel makers
Drug makers	Manganese workers
Dyers	Match makers
Enamel makers	Metal refiners
Feed additive makers	Organic chemical synthesizers
Ferromanganese alloy makers	Paint makers
Fertilizer makers	Permanganate workers
Fireworks makers	Rubber makers
Glass makers	Textile fiber bleachers
Hydroquinone makers	Textile printers
Ink makers	Varnish makers
Linoleum makers	Water treaters
Manganese alloy makers	Welders, electric arc
Manganese ore crushers	Wood preservative workers

### References

BOLTON, N. E.; CAVENDER, J. D., AND STACK, V. T., JR.: Determination of manganese in biological specimens. *Am. Indust. Hyg. Assoc. J.* 23: 319, 1962.

COTZIAS, G. C.: Manganese in health and disease. *Physiol. Rev.* 38: 503, 1958.

DAVIES, T. A. L.: Manganese pneumonitis. *Brit. J. Indust. Med.* 3: 111, 1946.

FLINN, R. H.; NEAL, P. A.; REINHART, W. H.; DALLA VALLE, J. M.; FULTON, W. B., AND DOOLEY, A. E.: Chronic manganese poisoning in an ore-crushing mill. *Pub. Health Bull.* No. 247. U.S. Government Printing Office, Washington, D.C., 1940.

PENALVER, R.: Manganese poisoning. *Indust. Med. & Surg.* 24: 1, 1955.

RODIER, J.: Manganese poisoning in Moroccan miners. *Brit. J. Indust. Med.* 12: 21, 1955.

TEPPER, L. B.: Hazards to health; manganese. *New Eng. J. Med.* 264: 347, 1961.

### (113) Mercaptans

The mercaptans include butyl mercaptan (butanethiol), ethyl mercaptan (ethanethiol, ethyl sulfhydrate), methyl mercaptan (methanethiol), and perchloromethyl mercaptan.

#### *Harmful Effects*

*Local* Contact dermatitis from primary irritation by liquid.

*Route of Entry* Inhalation of vapor.

*Systemic* In acute exposures, mercaptans have a narcotic effect and produce headache, nausea, vomiting, dizziness, and unconsciousness. Strong and disagreeable odors normally prevent overexposure.

#### *Special Diagnostic Test*

None.

#### *Recommended Threshold Limit*

*Butyl mercaptan*, 10 parts per million parts of air by volume or 35 milligrams per cubic meter of air.

*Ethyl mercaptan*, 20 parts per million parts of air by volume or 52 milligrams per cubic meter of air.

*Methyl mercaptan*, 20 parts per million parts of air by volume or 40 milligrams per cubic meter of air.

*Perchloromethyl mercaptan*, 0.1 part per million parts of air by volume or 0.8 milligram per cubic meter of air.

#### *Potential Occupational Exposures*

Dye makers	Motor fuel blenders
Fumigant makers	Organic chemical synthesizers
Fumigators	Rubber makers
Mercaptan workers	Skunk trappers
Methionine makers	Warning agent workers

#### *Reference*

FAIRCHILD, E. J. AND STOKINGER, H. E.: Toxicologic studies on organic sulfur compounds. 1, Acute toxicity of some aliphatic and aromatic thiols (mercaptans). *Am. Indust. Hyg. Assoc. J.* 19: 171, 1958.

## (114) Mercury and Compounds

*metallic mercury: quicksilver, hydrargyrum**Harmful Effects*

**Local** Certain mercurial compounds are primary skin and mucous membrane irritants. Allergic hypersensitization is seen less frequently.

**Routes of Entry** Inhalation of vapor. Percutaneous absorption of metal and organic compounds.

**Systemic** Acute severe exposures may produce abdominal pain, vomiting, diarrhea, gingivitis, pneumonitis, renal damage, and circulatory or respiratory failure.

Chronic excessive exposure to many inorganic mercury compounds may result in one or more of the three classical signs of gingivitis, tremor, and emotional instability. Headaches, insomnia, digestive disturbances, renal damage, hearing impairment, restriction of visual fields, and crystalline lens discoloration have also been described.

Toxicity resulting from exposure to certain organic mercurials, such as diethyl mercury and methyl mercury iodide, can often be differentiated from inorganic mercury toxicity. This condition is characterized by ataxia, tremor, dysarthria, impaired hearing, paresthesias, emotional instability, and restriction of visual fields.

Permanent sequelae may occur following either acute or chronic intoxication from inorganic or organic mercurial compounds.

*Special Diagnostic Test*

Analysis of urine for mercury. See Kopp and Keenan, 1963.

*Recommended Threshold Limit*

**Mercury**, 0.1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

**Mercury (organic compounds)**, 0.01 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Amalgam makers	Ceramic workers
Bactericide makers	Chlorine makers
Barometer makers	Dental amalgam makers
Battery makers, mercury	Dentists
Boiler makers	Direct current meter workers
Bronzers	Disinfectant makers
Calibration instrument makers	Disinfectors
Cap loaders, percussion	Drug makers
Carbon brush makers	Dye makers
Caustic soda makers	Electric apparatus makers

Electroplaters	Mercury workers
Embalmers	Miners, mercury
Explosive makers	Neon light makers
Farmers	Paint makers
Fingerprint detectors	Paper makers
Fireworks makers	Percussion cap makers
Fungicide makers	Pesticide workers
Fur preservers	Photographers
Fur processors	Pressure gage makers
Gold extractors	Refiners, mercury
Histology technicians	Seed handlers
Ink makers	Silver extractors
Insecticide makers	Switch makers, mercury
Investment casting workers	Tannery workers
Jewelers	Taxidermists
Laboratory workers, chemical	Textile printers
Lamp makers, fluorescent	Thermometer makers
Lamp makers, mercury arc	Vinyl chloride makers
Manometer makers	Wood preservative workers

### References

BATTIGELLI, M. C.: Mercury toxicity from industrial exposure. A critical review of the literature. *J. Occup. Med.* 2: 337 and 394, 1960.

GOLDWATER, L. J.; JACOBS, M. B., AND LADD, A. C.: Absorption and excretion of mercury in man. I, Relationship of mercury in blood and urine. *Arch. Environ. Health* 5: 537, 1962.

GRIEVE, W. T. AND WARD, W. M.: Report on organic mercury hazard to personnel involved in the testing and grading of seed grains. *Occup. Health Rev.* (Ottawa) 14(3): 14, 1962.

KOPP, J. F. AND KEENAN, R. G.: Determination of submicrogram quantities of mercury in urine by ion exchange separation. *Am. Indust. Hyg. Assoc. J.* 24: 1, 1963.

KURLAND, L. T.; FARO, S. N., AND SIEDLER, H.: Minamata disease; the outbreak of a neurologic disorder in Minamata, Japan, and its relationship to the ingestion of seafood contaminated by mercuric compounds. *World Neurology* 1: 370, 1960.

### (115) Methyl Alcohol

*methanol, carbinol, wood alcohol, wood spirit*

#### Harmful Effects

*Local* Contact with liquid can produce a dry, scaly, and fissured dermatitis. Both liquid and vapor irritate mucous membranes of eyes, nose, and throat.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid. *Systemic* Toxic effect of methyl alcohol on optic nerve is mediated through its oxidation product, formaldehyde, and may result in blurring of vision, pain in eyes, loss of central vision, or blindness. Other central

nervous system effects result from narcosis and include headache, nausea, giddiness, and loss of consciousness. Another oxidation product, formic acid, may produce acidosis. Severe intoxication may produce kidney and liver damage. Inhalation of vapor may irritate respiratory tract and produce bronchitis or broncho-pneumonia.

### *Special Diagnostic Tests*

Determination of methyl alcohol in blood, and methyl alcohol and formic acid in urine. Estimation of alkali reserve which may be impaired because of acidosis. *See Von Oettingen, 1958.*

### *Recommended Threshold Limit*

200 parts per million parts of air by volume or 260 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Acetic acid makers	Hectograph operators
Adhesive workers	Incandescent lamp makers
Alcohol distillery workers	Ink makers
Alcohol lamp users	Japan makers
Aldehyde pumpmen	Japanners
Antifreeze workers	Jet fuel workers
Art glass workers	Lacquerers
Automobile painters	Lacquer makers
Aviation fuel handlers	Lasters
Aviation fuel makers	Leather workers
Bookbinders	Linoleum makers
Bronzers	Lithographers
Brush makers	Metal polishers
Denatured alcohol workers	Methyl acrylate makers
Dimethyl sulfate makers	Methyl alcohol workers
Drug makers	Methyl amine makers
Dry cleaners	Methylation workers
Dye makers	Methyl bromide makers
Dyers	Methyl chloride makers
Ester makers	Methyl methacrylate makers
Explosive workers	Millinery workers
Feather workers	Motor fuel blenders
Felt hat makers	Organic chemical synthesizers
Flower makers, artificial	Painters
Formaldehyde makers	Paint makers
Foundry workers	Paint remover workers
Furniture polishers	Patent leather makers
Gilders	Perfume makers
Glass makers, safety	Photoengravers

Photographic film makers	Shoe stitchers
Polish makers	Soap makers
Printers	Solvent workers
Rayon makers	Straw hat makers
Resin makers	Sugar refiners
Rocket fuel handlers	Textile printers
Rocket fuel makers	Type cleaners
Rubber shoe cementers	Upholsterers
Rubber workers	Vacuum tube makers
Shellackers	Varnish workers
Shellac makers	Vulcanizers
Shoe factory workers	Wood alcohol distillers
Shoe finishers	Wood stainers
Shoe heel coverers, wood	Wood stain makers

### References

KEENEY, A. H. AND MELLINKOFF, S. M.: Methyl alcohol poisoning. *Ann. Int. Med.* 34: 331, 1951.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(116) Methyl Bromide (Bromomethane). *See* Bromine and Compounds

(117) Methyl Butyl Ketone. *See* Ketones

(118) Methyl Chloride

*monochloromethane, chloromethane*

### Harmful Effects

*Local* Evaporation of liquid from skin produces frostbite. Liquid also damages eye.

*Route of Entry* Inhalation of gas or vapor.

*Systemic* Methyl chloride acts as narcotic and also damages liver, kidneys, bone marrow, and central nervous system. Central nervous system effects are characteristically delayed and include headache, dizziness, vomiting, blurred or double vision, mental confusion, drowsiness, convolution, unconsciousness, and death. Recovery may be erratic.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

100 parts per million parts of air by volume or 210 milligrams per cubic meter of air.

## Potential Occupational Exposures

Aerosol packagers	Petroleum refinery workers
Drug makers	Polystyrene foam makers
Flavor extractors	Refrigeration workers
Low temperature polymerization workers	Rubber makers
Low temperature solvent workers	Silicone makers
Methylation workers	Thermometer makers, vapor pressure
Methyl cellulose makers	Thermometric equipment makers
Organic chemical synthesizers	Thermostatic equipment makers

## References

HANSEN, H.; WEAVER, N. K., AND VENEABLE, F. S.: Methyl chloride intoxication. Report of fifteen cases. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 328, 1953.  
 MACKIE, I. J.: Methyl chloride intoxication. *Med. J. Australia* 1: 203, 1961.

## (119) Methyl Chloroform

### *1,1,1-trichloroethane*

## Harmful Effects

**Local** Liquid and high vapor concentrations will irritate eyes on contact. Repeated skin contact will produce a dry, scaly, and fissured dermatitis.

**Route of Entry** Inhalation of vapor.

**Systemic** Narcotic effects of dizziness, incoordination, drowsiness, and unconsciousness have been produced by acute exposure to vapor concentrations approaching 1,000 p.p.m. If the worker is not removed after he has been overcome, death can result from respiratory failure or possibly ventricular arrhythmia. Fatty degeneration of liver occurred in laboratory animals undergoing chronic exposure to high concentrations. In human subjects transient elevation of urinary urobilinogen has been noted following exposure to anesthetic concentrations.

## Special Diagnostic Test

Infrared analysis of blood for 1,1,1-trichloroethane. See Stewart et al., 1961.

## Recommended Threshold Limit

350 parts per million parts of air by volume or 1,900 milligrams per cubic meter of air.

## Potential Occupational Exposures

Dry cleaners	Metal degreasers
Machinery cleaners	Stain removers

## References

STEWART, R. D.; GAY, H. H.; ERLEY, D. S.; HAKE, C. L., AND SCHAFER, A. W.: Human exposure to 1,1,1-trichloroethane vapor; relationship of expired air and blood concentrations to exposure and toxicity. *Am. Indust. Hyg. Assoc. J.* 22: 252, 1961.

TORKELSON, T. R.; OYEN, F.; MCCOLLISTER, D. D., AND ROWE, V. K.: Toxicity of 1,1,1-trichloroethane as determined on laboratory animals and human subjects. *Am. Indust. Hyg. Assoc. J.* 19: 353, 1958.

### (120) Methylene Chloride

*dichloromethane, methylene dichloride, methylene bichloride*

#### Harmful Effects

*Local* Repeated contact with this solvent will cause a dry, scaly, and fissured dermatitis. Liquid and vapor are irritating to eyes and upper respiratory tract.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Methylene chloride acts as narcotic in high concentrations, causing headache, nausea, vomiting, drowsiness, incoordination, paresthesias, and coma. High concentrations may also produce bronchitis and pulmonary edema.

#### Special Diagnostic Test

None.

#### Recommended Threshold Limit

500 parts per million parts of air by volume or 1,750 milligrams per cubic meter of air.

#### Potential Occupational Exposures

Aerosol packagers	Fumigators
Alkaloid processors	Lacquerers
Anesthetic makers	Lacquer workers
Bitumen processors	Leather finish workers
Cellulose acetate workers	Methylene chloride workers
Cellulose ester workers	Oil extractors
Cellulose ether workers	Oil processors
Crude rubber workers	Organic chemical synthesizers
Degreasers	Paint remover workers
Dentists	Perfume makers
Drug makers	Photographic film makers
Dye makers	Refrigeration workers
Fat extractors	Resin makers
Fire extinguisher workers	Rubber workers
Flavoring makers	Solvent workers
Fumigant makers	Stain removers

Textile finishers  
Varnish remover workers

Wax makers  
Wax removers

## References

IRISH, D. D.: Common chlorinated aliphatic hydrocarbon solvents. *Arch. Environ. Health* 4: 320, 1962.

MOSKOWITZ, S. AND SHAPIRO, H.: Fatal exposure to methylene chloride vapor. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 116, 1952.

### (121) Methyl Formate

*methyl methanoate*

## Harmful Effects

*Local* High concentrations are irritating to mucous membranes of upper respiratory tract. Repeated contact can produce a dry, scaly, and fissured dermatitis.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Vapors of methyl formate may have a narcotic effect. Systemic toxicity in industry is unusual.

## Special Diagnostic Test

None.

## Recommended Threshold Limit

100 parts per million parts of air by volume or 250 milligrams per cubic meter of air.

## Potential Occupational Exposures

Cellulose acetate workers  
Fumigant makers  
Fumigators  
Grain fumigators

Methyl formate workers  
Organic chemical synthesizers  
Pesticide workers  
Tobacco fumigators

## References

VON OETTINGEN, W. F.: The aliphatic acids and their esters: toxicity and potential dangers; the saturated monobasic aliphatic acids and their esters. *A.M.A. Arch. Indust. Health* 20: 517, 1959.

### (122) Methyl Mercaptan. *See* Mercaptans

### (123) Molybdenum and Compounds

## Harmful Effects

*Local* Unknown.

*Route of Entry* Inhalation of dust or fume.

*Systemic* No human cases of industrial toxicity following exposure to molybdenum or its compounds have been reported. Animal studies with

molybdenite, molybdic oxide, ammonium molybdate, calcium molybdate, and metallic molybdenum indicate a low order of toxicity. Available information concerning chronic exposure to molybdenum compounds is insufficient to define a health hazard.

### *Special Diagnostic Test*

Analysis of blood and urine for molybdenum. *See* Fairhall, 1957.

### *Recommended Threshold Limits*

*Molybdenum (soluble compounds)*, 5 milligrams per cubic meter of air.  
*Molybdenum (insoluble compounds)*, 15 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Ceramic makers	Molybdenum ore miners
Coal technologists	Molybdenum ore smelters
Drug makers	Molybdenum sheet makers
Dye makers	Molybdenum steel workers
Electric arc welders	Molybdenum wire makers
Electroplaters	Molybdenum workers
Ferroalloy workers	Petroleum refinery workers
Fertilizer makers	Pigment makers
Glass makers	Steel alloy makers
Lubricant makers	Tannery workers
Metal platers	Vacuum tube makers
Molybdenum iron workers	

### *References*

FAIRHALL, L. T.; DUNN, R. C.; SHARPLESS, N. E., AND PRITCHARD, E. A.: The toxicity of molybdenum. *Pub. Health Bull.* No. 293. U.S. Government Printing Office, Washington, D.C., 1945.

FAIRHALL, L. T.: *Industrial Toxicology*. 2nd ed. Williams & Wilkins Co., Baltimore, 1957.

## (124) Naphtha

*Petroleum naphtha* (ligroin, benzine, petroleum ether, petroleum benzine)  
*Coal tar naphtha* (hi-flash naphtha)

Certain petroleum naphthas contain varying amounts of benzene. It is known that a potential benzene hazard is associated with the use of such naphthas. *See* Benzene.

### *Harmful Effects*

*Local* The naphthas are irritating to skin, conjunctiva, and mucous membranes of upper respiratory tract.

**Routes of Entry** Inhalation of vapor. Percutaneous absorption of liquid is probably not important in development of systemic illness.

**Systemic** The naphthas may produce symptoms and signs of central nervous system depression similar to those resulting from gasoline intoxication. Coal tar naphtha, a mixture of aromatic hydrocarbons, including toluene, xylene, and pseudocumene has a greater propensity to produce toxicity than petroleum naphtha, consisting principally of a mixture of paraffin hydrocarbons.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

*Naphtha (petroleum)*, 500 parts per million parts of air by volume or 2,000 milligrams per cubic meter of air.

*Naphtha (coal tar)*, 200 parts per million parts of air by volume or 800 milligrams per cubic meter of air.

### Potential Occupational Exposures

Detergent makers	Photographic chemicals makers
Dry cleaners	Rubber coaters
Fat processors	Rubber makers
Insecticide workers	Solvent workers
Laboratory workers, chemical	Stainers
Metal degreasers	Stain makers
Naphtha workers	Varnish makers
Oil processors	Wax makers
Painters	Wool processors
Paint makers	Xylene makers
Petroleum refinery workers	

### References

ELKINS, H. B.; COMPRONI, E. M., AND PAGNOTTO, L. D.: Industrial benzene exposure from petroleum naphtha. 2, Pertinent physical properties of hydrocarbon mixtures. *Am. Indust. Hyg. Assoc. J.* 24: 99, 1963. Consideration should be given to the benzene exposure possibly resulting from the use of petroleum naphtha containing as little as 2 percent benzene by weight.

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons*. Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

PAGNOTTO, L. D.; ELKINS, H. B.; BRUGSCH, H. G., AND WALKLEY, J. E.: Industrial benzene exposure from petroleum naphtha. 1, Rubber coating industry. *Am. Indust. Hyg. Assoc. J.* 22: 417, 1961. Benzene in certain naphtha solvents varies up to 9.3 percent by weight. Saturators, using naphthas containing more than 3 percent benzene, showed excessive benzene exposure; for churn men the exposure was lower and for spreaders lower than that for churn men.

## (125) Naphthalene

*naphthalin, naphthene, moth flakes, tar camphor, white tar*

*Harmful Effects*

*Local* Contact dermatitis from primary irritation or allergic hypersensitivity, or both. Eye irritation from vapors.

*Route of Entry* Inhalation of vapor or dust.

*Systemic* High concentrations can produce headache, nausea, vomiting, profuse perspiration, optic neuritis, and hematuria. Prolonged exposure to high concentrations can produce opacity of lens.

*Special Diagnostic Tests*

Determination of naphthalene in urine and blood. Heinz bodies may be seen in erythrocytes. See Von Oettingen, 1958.

*Recommended Threshold Limit*

(Tentative) 10 parts per million parts of air by volume or 50 milligrams per cubic meter of air.

*Potential Occupational Exposures*

o-Aminobenzoic acid makers	Lampblack makers
Beta naphthol makers	Lubricant workers
Celluloid makers	Moth repellent workers
Coal tar workers	Naphthalene workers
Cutting fluid workers	Phthalic anhydride makers
Dye chemical makers	Resin makers
Dye intermediate makers	Scintillation counter makers
Fumigant workers	Smokeless powder makers
Fungicide makers	Soil treaters
Hydronaphthalene makers	Tannery workers
Insecticide workers	Textile chemical makers

*References*

GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons*. Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (126) Naphthylamine (Beta)

Pennsylvania was the first State to adopt a regulation for the control of this material. On October 27, 1961 the following section was adopted and made a part of Chapter 4, Article 434 (Regulations for Control of Dangerous Materials) of the Rules and Regulations of the Commonwealth of Pennsylvania Department of Health:

### Section 1. Beta-naphthylamine

No person, corporation, partnership or association shall manufacture, use or permit to be used, store, transport, or otherwise handle beta-naphthylamine. Any area where beta-naphthylamine has been used, stored, or otherwise handled shall be decontaminated to assure that no individual shall be exposed.

### Harmful Effects

**Local** Beta-naphthylamine is mildly irritating to skin and has produced contact dermatitis.

**Routes of Entry** Inhalation of dust and percutaneous absorption.

**Systemic** A metabolite, the 1-hydroxy derivative of beta-naphthylamine, is a potent carcinogen. The metabolite acts on urinary bladder mucosa causing cystitis and papillomata which may become malignant. Symptoms are frequent urination, dysuria, and hematuria, which appear after several years of exposure or several years after last exposure. Alpha-naphthylamine is unimportant toxicologically except for its frequent contamination by beta-naphthylamine.

### Special Diagnostic Test

Analysis of urine for naphthylamine. See Von Oettingen, 1958.

### Recommended Threshold Limit

Not established.

### Potential Occupational Exposure

Dye makers	beta-Naphthylamine workers
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### References

CASE, R. A. M.; HOSKER, M. E.; MC DONALD, D. B., AND PEARSON, J. T.: Tumors of the urinary bladder in workmen engaged in the manufacture and use of certain dyestuff intermediates in the British chemical industry. I, The role of aniline, benzidine, alpha-naphthylamine, and beta-naphthylamine. *Brit. J. Indust. Med.* 11: 75, 1954.

VIGLIANI, E. C. AND BARSOTTI, M.: Environmental tumors of the bladder in some Italian dyestuff factories. *Med. Lavoro* 52: 241, 1961.

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (127) Natural Gas

Natural gas consists primarily of methane (85 percent) with lesser amounts of ethane (9 percent), propane (3 percent), nitrogen (2 percent), and butane (1 percent).

*Harmful Effects**Local* None.*Route of Entry* Inhalation of gas.*Systemic* Displacement of air by the gas may lead to shortness of breath, unconsciousness, and death from hypoxemia. A mild central nervous system depressant effect has been attributed to the homologs of methane in the gas. Incomplete combustion may result in production of carbon monoxide.*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Acetaldehyde makers	Hydrogenated oil makers
Acetylene makers	Hydrogen makers
Ammonia makers	Methanol makers
Carbon black makers	Natural gas workers
Coal miners	Nitric acid makers
Ethanol makers	Organic chemical synthesizers
Formaldehyde makers	Petroleum refinery workers
Gas fuel users	Power plant workers, electric
Helium extractors	Synthesis gas makers
Hydrocarbon fuel makers	Vinyl chloride makers

*Reference*

HENSON, E. V.: Toxicology of some of the aliphatic and alicyclic hydrocarbons. *J. Occup. Med.* 1: 105, 1959.

## (128) Nickel and Compounds

*Harmful Effects**Local* Nickel salts produce allergic contact dermatitis. A type of dermatitis referred to as *nickel itch* may be seen in nickel miners, smelters, and refiners. This condition is characterized by an erythematous, papular, pruritic rash, often beginning in web of fingers and spreading to fingers, wrists, and forearms.*Route of Entry* Inhalation of dust or fume.*Systemic* Nickel carbonyl is thought to be the most toxic of nickel compounds. See Carbonyls. Metallic nickel and its salts are considered to be of very low level of toxicity when taken into body. There has been reported an increase in incidence of cancer of lung and ethmoid sinuses in men exposed to dust in nickel refining.

## Special Diagnostic Tests

Analysis of blood and urine for nickel. See Kincaid et al., 1956.

## Recommended Threshold Limit

(Nickel carbonyl) 0.001 part per million parts of air by volume or 0.007 milligram per cubic meter of air.

## Potential Occupational Exposures

Battery makers, storage	Nickel refiners
Cemented carbide makers	Nickel smelters
Ceramic makers	Nickel workers
Disinfectant makers	Oil hydrogenators
Dyers	Organic chemical synthesizers
Electroplaters	Paint makers
Enamelers	Pen point makers
Gas mask makers	Petroleum refinery workers
Ink makers	Spark plug makers
Jewelers	Steel makers, stainless
Magnet makers	Textile dyers
Mond process workers	Vacuum tube makers
Nickel alloy makers	Varnish makers
Nickel miners	

## References

DOLL, R.: Cancer of the lung and nose in nickel workers. *Brit. J. Indust. Med.* 15: 217, 1958.

KINCAID, J. F.; STANLEY, E. L.; BECKWORTH, C. H., AND SUNDERMAN, F. W.: Nickel poisoning. 3, Procedures for detection, prevention, and treatment of nickel carbonyl exposure including a method for the determination of nickel in biologic materials. *Am. J. Clin. Path.* 26: 107, 1956.

MORGAN, J. G.: Some observations on the incidence of respiratory cancer in nickel workers. *Brit. J. Indust. Med.* 15: 224, 1958.

SUNDERMAN, F. W. AND KINCAID, J. F.: Nickel poisoning. 2, Studies on patients suffering from acute exposure to vapors of nickel carbonyl. *J. Am. Med. Assoc.* 155: 889, 1954.

SUNDERMAN, F. W. AND SUNDERMAN, F. W., JR.: Loeffler's syndrome associated with nickel sensitivity. *Arch. Int. Med.* 107: 405, 1961.

## (129) Nickel Carbonyl

### *nickel tetracarbonyl*

## Harmful Effects

*Local* Contact dermatitis, possibly allergic, has been reported.

*Route of Entry* Inhalation of vapor.

*Systemic* Acute intoxication from nickel carbonyl vapor is due to irritation of lungs and to toxic effect on central nervous system. Early effects following acute exposure consist of headache, giddiness, unsteady

gait, nausea, vomiting, and a dry cough. A latent period of several hours to days may follow initial symptoms. Delayed symptoms of retrosternal pain, chest tightness, cough, dyspnea, extreme weakness, convulsions, hallucinations, delirium, nausea, and vomiting may terminate with pulmonary edema and respiratory or circulatory failure. Long exposure to low concentrations of nickel carbonyl is suspected of causing an increased incidence of carcinoma of respiratory tract including nasal sinuses.

### *Special Diagnostic Tests*

Analysis of blood and urine for nickel. *See* Sunderman and Kincaid, 1954.

### *Recommended Threshold Limit*

0.001 part per million parts of air by volume or 0.007 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Foundry workers

Nickel carbonyl workers

Gas platers

Petroleum refinery workers

Mond process workers

### *References*

HUEPER, W. C.: Carcinogens in the human environment. *Arch. Path.* 71: 237, 1961.  
 SUNDERMAN, F. W. and KINCAID, J. F.: Nickel poisoning. 2, Studies on patients suffering from acute exposure to vapors of nickel carbonyl. *J. Am. Med. Assoc.* 155: 889, 1954.

(130) Nicotine. *See* Pesticides Section

(131) Nitric Acid

*aqua fortis, hydrogen nitrate*

### *Harmful Effects*

*Local* Very corrosive. Capable of producing severe burns, ulcers and necrosis of skin, mucous membranes and eyes. Prolonged exposure to vapor may cause yellowing of skin and erosion of teeth.

*Route of Entry* Inhalation of vapor.

*Systemic* Inhalation may cause irritation of entire respiratory tract. Pulmonary edema may result. Pulmonary fibrosis has been reported to follow inhalation.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

10 parts per million parts of air by volume or 25 milligrams per cubic meter of air.

## Potential Occupational Exposures

Aircraft workers	Lithographers
Ammonium nitrate makers	Mirror makers
Bleachers	Nitration workers
Brass cleaners	Nitric acid workers
Bright-dip workers	Nitrobenzene makers
Cellulose nitrate makers	Nitro-compound workers
Drug makers	Ore flotation workers
Dye makers	Organic chemical synthesizers
Electroplaters	Photoengravers
Etchers	Rocket fuel handlers
Explosive makers	Rock phosphate acidulators
Jewelers	Steel etchers
Laboratory workers, chemical	Sulfuric acid makers

### (132) Nitrobenzene

*nitrobenzol, essence of mirbane, oil of mirbane, oil of bitter almonds*

#### Harmful Effects

*Local* Nitrobenzene may produce contact dermatitis through primary irritation or allergic hypersensitization.

*Route of Entry* Inhalation of vapor or percutaneous absorption of liquid.

*Systemic* Nitrobenzene converts hemoglobin to methemoglobin, resulting in headache, dizziness, shortness of breath, and a bluish-discoloration of the lips (blue-lip), mucous membranes, and skin. Central nervous system depression, anemia, and liver damage may occur with acute or chronic intoxication.

#### Special Diagnostic Tests

Analysis of urine for nitrophenol, and of blood for methemoglobin. See Salmowa et al., 1963, and Von Oettingen, 1941.

#### Recommended Threshold Limit

1 part per million parts of air by volume or 5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

## Potential Occupational Exposures

Aniline makers	Organic chemical synthesizers
Azobenzene makers	Petroleum refinery workers
Benzidine makers	Quinoline makers
Explosive workers	Shoe polish makers
Glue makers	Soap makers
Ink makers	Stainers
Lacquer makers	Stain makers
Metal polish makers	Vanillin makers
Nitrobenzene workers	

## References

SALMOWA, J.; PIOTROWSKI, J., AND NEUHORN, U.: Evaluation of exposure to nitrobenzene; absorption of nitrobenezene vapour through lungs and excretion of p-nitrophenol in urine. *Brit. J. Indust. Med.* 20: 41, 1963.

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

(133) Nitroethane. *See* Nitroparaffins

(134) Nitrogen Oxides

The most common oxides of nitrogen include nitrous oxide ( $N_2O$ , di-nitrogen monoxide), nitric oxide ( $NO$ , nitrogen monoxide), nitrogen dioxide ( $NO_2$ ) which usually consists of an equilibrium mixture of nitrogen dioxide and nitrogen tetroxide ( $N_2O_4$ , dinitrogen tetroxide). Nitrogen trioxide ( $N_2O_3$ , dinitrogen trioxide) dissociates into nitric oxide and nitrogen dioxide. Nitrogen pentoxide ( $N_2O_5$ , dinitrogen pentoxide), upon contact with air, decomposes into nitrogen dioxide and oxygen.

(1) Nitrous Oxide

*dinitrogen monoxide, nitrogen monoxide, factitious air, hyponitrous acid anhydride, laughing gas*

## Harmful Effects

*Local* None.

*Route of Entry* Inhalation of gas.

*Systemic* Mild anesthetic action.

## Special Diagnostic Test

None.

## Recommended Threshold Limit

Not established.

## Potential Occupational Exposures

Aerosol packagers, food	Nitrous oxide workers
Dental technicians	Nurses
Dentists	Physicians
Medical technicians	Rocket fuel makers

(2) Nitric Oxide

At ordinary temperatures, nitric oxide combines with atmospheric oxygen to form nitrogen dioxide.

## (3) Nitrogen Tetroxide

*dinitrogen tetroxide*

**Nitrogen tetroxide** is an equilibrium mixture of nitrogen tetroxide and nitrogen dioxide.

*Harmful Effects*

**Local** Nitrogen tetroxide is an extremely corrosive liquid and may cause severe burns.

**Route of Entry** Inhalation of gas.

**Systemic** Effects are due to inhalation of the emitted nitrogen dioxide gas.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

(Nitrogen dioxide) 5 parts per million parts of air by volume or 9 milligrams per cubic meter of air.

*Potential Occupational Exposure*

Nitrogen tetroxide workers	Rocket fuel makers.
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## (4) Nitrogen Dioxide

*Harmful Effects*

**Local** Very irritating to eyes and mucous membranes. Prolonged low-level exposures may produce yellowish to brown staining of the teeth and skin.

**Route of Entry** Inhalation of gas.

**Systemic** Exposure to high concentrations may produce immediate coughing and chest pain. When lower concentrations are inhaled, there may be only mild signs of bronchial irritation followed by a 5- to 12-hour symptom free period. Subsequently, the onset of signs and symptoms of acute pulmonary edema may be noted. Death often results within 24 hours. If the acute episode is survived, sequelae such as bronchiectasis or emphysema may develop.

Methemoglobinemia, generally of a mild degree, may be produced by exposure to nitrous fumes containing small amounts of nitric oxide.

Nitrogen dioxide may be formed from fresh green silage in amounts which, when restrained in the confines of a silo, may constitute a serious health hazard. The name *silo-filler's disease* has been used to designate the lung condition caused by exposure to nitrogen dioxide evolved in this way.

The following events characterize this disease: Exposure to the gases contained within a recently filled silo has been followed almost immediately by malaise, cough, dyspnea, chest pain, chills, fever, nausea, and vomiting.

These signs and symptoms may last from several days to several weeks. In some cases, complete resolution may occur while in others there may be a progression to severe pulmonary insufficiency and death. A latent period of 2 or 3 weeks between the initial onset of signs and symptoms and the final progression to potentially fatal pulmonary insufficiency has been described.

The chest roentgenogram may reveal a picture varying from one of a diffuse, patchy, confluent infiltration to one of numerous, uniformly scattered, nodular densities ranging in size from 1 to 5 mm in diameter. These roentgenographic patterns may undergo partial or complete clearing as the disease resolves. However, in some cases, there may remain roentgenographic evidence of pulmonary fibrosis.

Pulmonary function studies done after recovery from the initial acute episode may reveal striking variations from the expected normal values. Findings suggestive of obstructive pulmonary emphysema have been reported. These changes include an increase in residual volume, a decreased maximum breathing capacity, and some prolongation of the timed vital capacity. Changes in the diffusion capabilities of the lung do not seem to be significant.

Histologic sections of biopsy or necropsy specimens of lung tissue acquired during the initial acute stages of disease usually indicate extensive bronchopneumonia. During later stages, however, the histopathologic picture is frequently one of bronchiolitis fibrosa obliterans. No significant pathogenic microorganisms have been consistently identified in patients with this disease.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

5 parts per million parts of air by volume or 9 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Brass cleaners	Nitric acid makers
Braziers	Nitrogen dioxide workers
Bright-dip workers	Oxidized cellulose (cellulosic acid) compound makers
Bronze cleaners	Pipe fitters
Copper cleaners	Plasma torch operators
Cotton bleachers	Raw silk bleachers
Electric arc welders	Rocket fuel makers
Electroplaters	Silo fillers
Flour bleachers	Sulfuric acid makers
Nitrate workers	

## References

CLANCY, P. J.; WATSON, S. L., AND REARDAN, J. B.: Nitrogen tetroxide exposure in the missile industry. *J. Occup. Med.* 4: 691, 1962.

GRAYSON, R. R.: Silage gas poisoning; nitrogen dioxide pneumonia, a new disease in agricultural workers. *Ann. Int. Med.* 45: 393, 1956.

LA FLECHE, L. R.; BOLVIN, C., AND LEONARD, C.: Nitrogen dioxide, a respiratory irritant. *Canad. Med. Assoc. J.* 84: 1438, 1961.

LEIB, G. M. P.; DAVIS, W. N.; BROWN, T., AND MC QUIGGAN, M.: Chronic pulmonary insufficiency secondary to silo-filler's disease. *Am. J. Med.* 24: 471, 1958.

LOWRY, T. AND SCHUMAN, L. M.: "Silo fillers disease," a syndrome caused by nitrogen dioxide. *J. Am. Med. Assoc.* 162: 153, 1956.

OFFICE OF DIRECTOR, DEFENSE RESEARCH AND ENGINEERING, DEPARTMENT OF DEFENSE: *The Handling and Storage of Liquid Propellants*. U.S. Government Printing Office, Washington, D.C., 1961.

RAFIL, S. AND GODWIN, M. C.: Silo filler's disease; relapse following latent period. *Arch. Path.* 72: 424, 1961.

### (135) Nitroglycerin

*nitroglycerol, glyceryl trinitrate, trinitroglycerol*

## Harmful Effects

*Local* May cause contact dermatitis because of allergic hypersensitivity or primary irritation or both.

*Route of Entry* Inhalation of dust or vapor; ingestion of dust; percutaneous absorption of liquid.

*Systemic* Powerful vasodilatation with resultant flushing of skin and throbbing headache. Blood pressure may be lowered. Visual acuity may be diminished, or total temporary blindness may occur. Methemoglobin is formed but only in small amounts. Transitory mental aberration may occur. Massive exposures may cause loss of consciousness due to peripheral vascular dilatation. It is uncertain as to whether long-term exposure to nitroglycerin has an adverse effect upon the cardiovascular system. Toxic effects of nitroglycerin are accentuated by alcohol ingestion.

## Special Diagnostic Test

None.

## Recommended Threshold Limit

(Nitroglycerin with ethylene glycol dinitrate) 0.2 part per million parts of air by volume or 2 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

## Potential Occupational Exposures

Drug makers

Rocket fuel makers

Dynamite makers

Shell fillers

Missile technicians

Smokeless powder makers

Nitroglycerin workers

## References

BARSOTTI, M.: Attacks of stenocardia in workers engaged in the production of dynamites with nitroglycol. *Med. Lavoro* 45: 544, 1954.

FORSSMAN, S.; MASRELIEZ, N.; JOHANSSON, G.; SUNDELL, G.; WILANDER, O., AND BOSTROM, G.: The health of workers with nitro-compounds in three Swedish explosives factories. *Arch. Gewerbeopath.* 16: 157, 1958.

LAWS, G. C.: The effects of nitroglycerin upon those who manufacture it. *J. Am. Med. Assoc.* 31: 793, 1898.

RABINOWITCH, I. M.: Acute nitroglycerin poisoning. *Canad. Med. Assoc. J.* 50: 199, 1944.

VON OETTINGEN, W. F.: The effects of aliphatic nitrous and nitric acid esters on the physiological functions with special reference to their chemical constitution. *Nat. Inst. Health Bull.* No. 186. U.S. Government Printing Office, Washington, D.C., 1946.

YEE, H. T.; FOSDICK, L. B., AND BOURNE, H. G., JR.: Nitroglycerin and nitroglycol exposure in an explosives plant. *Am. Indust. Hyg. Assoc. J.* 20: 45, 1959.

(136) Nitromethane. *See* Nitroparaffins

(137) Nitroparaffins

The nitroparaffins include nitromethane, nitroethane, and nitropropane.

### Harmful Effects

*Local* The nitroparaffins are mild irritants to eyes and upper respiratory tract. Nitromethane is mildly irritating to skin.

*Route of Entry* Inhalation of vapor.

*Systemic* High concentrations may produce light narcosis and irritation of central nervous system. Liver and kidney damage have been observed in animal experiments. Only one notable industrial exposure has been reported, with symptoms of anorexia, nausea, vomiting, diarrhea, and occipital headache from inhalation of 2-nitropropane.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

*Nitromethane*, 100 parts per million parts of air by volume or 250 milligrams per cubic meter of air.

*Nitroethane*, 100 parts per million parts of air by volume or 310 milligrams per cubic meter of air.

*2-Nitropropane*, 25 parts per million parts of air by volume or 90 milligrams per cubic meter of air. Same value for 1-Nitropropane.

### Potential Occupational Exposures

Alkyd resin makers

Cellulose acetopropionate workers

Artificial resin makers

Dye makers

Cellulose acetate workers

Fat processors

Cellulose acetobutyrate workers

Nitrocellulose workers

Nitroethane workers	Stainers
Nitromethane workers	Stain makers
Nitropropane workers	Vinyl resin makers
Organic chemical synthesizers	Wax makers
Rocket fuel makers	

### Reference

SKINNER, J. B.: Toxicity of 2-nitropropane. *Indust. Med.* 16: 441, 1947.

### (138) Nitrophenols (Ortho-, Meta- and Para-)

#### Harmful Effects

*Local* Unknown.

*Routes of Entry* Percutaneous absorption of liquid; inhalation of vapor.

*Systemic* No industrial cases of poisoning have been recorded. Exposure to vapors may produce methemoglobinemia. Paranitrophenol is the most toxic of the isomers.

#### Special Diagnostic Test

Analysis of urine for paranitrophenol. See Mountain et al., 1951.

#### Recommended Threshold Limit

Not established.

#### Potential Occupational Exposures

Aminophenol makers	Nitroanisole makers
Drug makers	Nitrophenol workers
Dye makers	Organic chemical synthesizers
Explosive makers	Photographic chemical makers
Fungicide makers	Textile makers
Indicator makers, chemical	

#### References

MOUNTAIN, J. T.; ZLOTOLOW, H., AND O'CONOR, G. T.: Determination of paranitrophenol in urine in parathion poisoning cases. *Indust. Health Monthly* (USPHS) 11: 88, (June) 1951.

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

### (139) Nitropropane. See Nitroparaffins

### (140) Osmium and Compounds

Metallic osmium is considered to be nontoxic.

#### Harmful Effects

*Local* Upon heating in air osmium produces osmium tetroxide fume (osmic acid) which is irritating to mucous membranes and eyes. Certain

osmium salts, the chloride for example, may exhibit a caustic effect upon skin.

*Route of Entry* Inhalation of vapor or fume.

*Systemic* Inhalation of osmium tetroxide fume has been reported to produce extreme lung irritation, frequently progressing to bronchopneumonia. Asthma-like symptoms have also been reported to occur following inhalation of vapors of certain osmium compounds.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

(Osmium tetroxide) 0.002 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Alloy makers	Organic chemical synthesizers
Ammonia makers, synthetic	Osmium workers
Electric contact makers	Pen point makers
Histology technicians	Phonograph needle makers
Incandescent lamp makers	Platinum hardeners
Machine bearing makers	

### *Reference*

MC LAUGHLIN, A. I. G.; MILTON, R., AND PERRY, K. M. A.: Toxic manifestations of osmium tetroxide. *Brit. J. Indust. Med.* 3: 183, 1946.

## (141) Oxalic Acid

*dicarboxylic acid, ethane-di-acid, ethanedioic acid*

### *Harmful Effects*

*Local* Corrosive action on skin and mucous membranes may produce ulceration.

*Route of Entry* Inhalation of mist.

*Systemic* The calcium-complexing action of oxalate depresses level of ionized calcium in body fluids, producing severe disturbances of heart as well as muscle twitching, cramps, and central nervous system depression. Renal injury is frequently produced by acute poisoning but is rarely the cause of death.

### *Special Diagnostic Test*

Determination of blood calcium levels. See Gleason et al., 1957.

### *Recommended Threshold Limit*

Not established.

## Potential Occupational Exposures

Automobile radiator cleaners	Paint remover makers
Bleachers	Paint removers
Bleach makers	Paper makers
Celluloid makers	Photoengravers
Ceramic makers	Photographic workers
Coal washers	Pigment makers
Cream of tartar makers	Rayon bleachers
Dextrin makers	Rubber makers
Drug makers	Rust remover makers
Dye makers	Rust removers
Dyers	Stain removers
Formic acid makers	Stearin makers
Glycerine makers	Straw hat bleachers
Hydrocyanic acid makers	Tannery workers
Ink makers	Tartaric acid makers
Ink remover makers	Textile dyers
Laundry workers	Textile printers
Leather bleachers	Varnish remover makers
Lithographers	Varnish removers
Metal polish makers	Wood bleachers
Methanol makers	Wood cleaners
Organic chemical synthesizers	Wood cleanser makers
Oxalic acid workers	

## Reference

GLEASON, M. N.; GOSELIN, R. E., AND HODGE, H. C.; *Clinical Toxicology of Commercial Products; Acute Poisoning (Home and Farm)*. Williams & Wilkins Co., Baltimore, 1957.

## (142) Ozone

### Harmful Effects

*Local* Irritant to eyes and mucous membranes.

*Route of Entry* Inhalation of gas.

*Systemic* Pulmonary edema and hemorrhage may result from severe exposure. Less severe exposure may produce headache, malaise, shortness of breath, and drowsiness.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

0.1 part per million parts of air by volume or 0.2 milligram per cubic meter of air.

*Potential Occupational Exposures*

Air treaters	Oil bleachers
Arc cutters	Organic chemical synthesizers
Arc welders, argon shielded	Ozone workers
Arc welders, electric	Photoengravers
Arc welders, heliarc	Photographers
Arc workers, electric	Plasma torch operators
Bactericide makers	Sewage gas treaters
Electroplaters	Textile bleachers
Food preservers (cold storage)	Ultraviolet lamp workers
Hydrogen peroxide makers	Water treaters
Industrial waste treaters	Wax bleachers
Odor controllers	

*Reference*

STOKINGER, H. E.: Ozone toxicity. A review of the literature through 1953. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 9: 367, 1954.

(143) Pentanone. *See* Ketones

(144) Perchloroethylene

*tetrachloroethylene, carbon dichloride, ethylene tetrachloride*

*Harmful Effects*

*Local* Repeated contact with liquid causes a dry, scaly, and fissured dermatitis. High concentrations produce eye and nose irritation.

*Routes of Entry* Inhalation of vapor; of lesser importance, percutaneous absorption of liquid.

*Systemic* Primary systemic effect is narcosis, with symptoms of headache, dizziness, nausea, incoordination, and somnolence. Repeated exposures to high concentrations can produce a mild hepatitis.

*Special Diagnostic Test*

Analysis of blood for tetrachloroethylene. *See* Stewart, Gay et al., 1961.

*Recommended Threshold Limit*

100 parts per million parts of air by volume or 670 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Cellulose ester processors	Dope processors
Cellulose ether processors	Drug makers (anthelmintics)
Degreasers	Dry cleaners
Detergent makers	Electroplaters

Fumigant workers	Rubber workers
Gum processors	Soap workers
Heat transfer workers	Solvent workers
Metal degreasers	Tar processors
Organic chemical synthesizers	Vacuum tube makers
Paraffin processors	Wax makers
Perchloroethylene workers	Wool scourers
Printers	

### References

COLER, H. R. AND ROSSMILLER, H. R.: Tetrachlorethylene exposure in a small industry. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 227, 1953.

QUERIES AND MINOR NOTES: Toxicity of tetrachloroethylene. *J. Am. Med. Assoc.* 131: 1468, 1946.

STEWART, R. D.; ERLEY, D. S.; SCHAFER, A. W., AND GAY, H. H.: Accidental vapor exposure to anesthetic concentrations of a solvent containing tetrachloroethylene. *Indust. Med. & Surg.* 30: 327, 1961.

STEWART, R. D.; GAY, H. H.; ERLEY, D. S.; HAKE, C. L., AND SCHAFER, A. W.: Human exposure to tetrachloroethylene vapor. Relationship of expired air and blood concentrations to exposure and toxicity. *Arch. Environ. Health* 2: 516, 1961.

(145) Perchloromethyl Mercaptan. *See* Mercaptans

### (146) Phenol

*carbolic acid, phenic acid, phenylic acid, phenyl hydrate, hydroxybenzene, monohydroxybenzene*

### Harmful Effects

*Local* A primary irritant possessing strong corrosive properties for all tissues of body.

*Route of Entry* Inhalation of mist or vapor; percutaneous absorption of mist, vapor, or liquid.

*Systemic* Acute poisoning is mainly characterized by central nervous system manifestations including tinnitus, vertigo, tremor, excitement, and convulsions. Pneumonia often follows. Chronic phenol poisoning is characterized by headache, fatigue, cough, anorexia, insomnia, nervousness, paresthesias, weight loss, and cachexia. Renal and hepatic damage frequently follow phenol intoxication.

### Special Diagnostic Tests

Analysis of urine and blood for phenol. *See* Von Oettingen, 1958.

### Recommended Threshold Limit

5 parts per million parts of air by volume or 19 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Battery makers, dry	Paint removers
Coal tar workers	Pentachlorophenol makers
Disinfectant makers	Perfume makers
Drug makers	Phenol workers
Dye makers	Photographic material workers
Dyers	Picric acid makers
Etchers	Resin makers
Explosive workers	Rubber reclaimers
Gas workers, illuminating	Rubber workers
Gas purifiers	Stillmen, carbolic acid
Herbicide makers	Surgical dressing makers
Lampblack makers	Textile printers
Lubricating oil processors	Varnish makers
Paint makers	Weed killers
Paint remover makers	Wood preservers

*References*

EVANS, S. J.: Acute phenol poisoning. *Brit. J. Indust. Med.* 9: 227, 1952.  
 VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(147) Phenylhydrazine  
*hydrazinobenzene*

*Harmful Effects*

*Local* Eczematous contact dermatitis from primary irritation as well as allergic sensitivity.

*Route of Entry* Inhalation of vapor or mist; percutaneous absorption.

*Systemic* Phenylhydrazine hemolyses red blood cells and is a slight methemoglobin former. It also causes injury to liver, kidneys, and heart. Systemic findings and symptoms include anemia, leukopenia, hepatitis, headache, weakness, dizziness, anorexia, gastritis, diarrhea, hematuria, and albuminuria.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

5 parts per million parts of air by volume or 22 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

*Potential Occupational Exposures*

Analytical chemists	Nitron makers
Antipyrine makers	Organic chemical synthesizers
Drug makers	Phenylhydrazine workers
Dye makers	

## Reference

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

### (148) Phosgene

*carbonyl chloride, carbon oxychloride, combat gas, D-Stoff, chloroformyl chloride*

#### Harmful Effects

*Local* Contact dermatitis from primary irritation, conjunctival and upper respiratory tract irritation.

*Route of Entry* Inhalation of gas.

*Systemic* Acute exposure produces pulmonary edema frequently preceded by a latent period of several hours' duration. Death may result from respiratory or cardiac failure.

#### Special Diagnostic Test

None.

#### Recommended Threshold Limit

1 part per million parts of air by volume or 4 milligrams per cubic meter of air.

#### Potential Occupational Exposures

Chlorinated compound makers

Organic chemical synthesizers

Dye makers

Phosgene workers

Firemen

Resin makers

Glass makers

### (149) Phosphine

*hydrogen phosphide, phosphoretted hydrogen*

#### Harmful Effects

*Local* High concentrations of gas are irritating to eyes, nose and skin.

*Route of Entry* Inhalation of gas.

*Systemic* Acute effects are secondary to central nervous system depression, irritation of lungs, and damage to kidneys and other organs and include weakness, fatigue, hypotension, bradycardia, headache, dizziness, fainting, drowsiness, thirst, abdominal pain, nausea, vomiting, diarrhea, dyspnea, bronchitis, pulmonary edema, albuminuria, hematuria, tremors, staggering gait, convulsions, coma and death. In chronic poisoning there may be disturbances in sight, speech, and motor functions and effects seen in chronic phosphorus poisoning such as nonhemolytic anemia, brittle teeth, and necrosis of the lower jaw.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

0.3 part per million parts of air by volume or 0.4 milligram per cubic meter of air.

*Potential Occupational Exposures*

Acetylene generator workers	Metal slag workers
Acetylene workers	Organic chemical synthesizers
Aluminum phosphide workers	Phosphine workers
Calcium carbide workers	Pyrites sulfuric acid workers
Cement workers	Rustproofers
Ferrosilicon workers	Sodium phosphide workers
Firemen	Sulfuric acid tank cleaners
Grain fumigators	Welders
Metal alloy workers	Zirconium diphosphide makers
Metal refiners	

*Reference*

HARGER, R. N. AND SPOLYAR, L. W.: Toxicity of phosphine, with a possible fatality from this poison. *A.M.A. Arch. Indust. Health* 18: 497, 1958.

### (150) Phosphorus and Compounds

Elemental phosphorus exists in two forms, a red form which is nontoxic, and a yellow or white which is very toxic. Frequently the red is contaminated with small amounts of the yellow.

*Harmful Effects*

*Local* Skin contact with yellow phosphorus results in production of severe burns. In addition, the following phosphorus compounds are reported to be potent irritants of skin, eyes, and mucous membranes of nose, throat, and respiratory tract:

Phosphorus trichloride	Phosphorus trisulfide
Phosphorus pentachloride	Phosphorus pentasulfide
Phosphorus oxychloride	Phosphorus sesquisulfide
Phosphorus tribromide	Phosphoric acid
Phosphorus pentabromide	

*Routes of Entry* Ingestion and percutaneous absorption of dust; inhalation of dust or fume.

*Systemic* Ingestion of yellow phosphorus produces severe poisoning, beginning with local gastrointestinal irritation, progressing to systemic poisoning. Shock may ensue rapidly. If death is not immediate, patient

may succumb later to liver, kidney, or heart failure brought about by direct action of phosphorus on these organs.

Inhalation of fumes produced by the phosphorus compounds listed above may cause irritation of pulmonary tissues with resultant acute pulmonary edema.

Chronic phosphorus poisoning is result of continued absorption of small amounts of yellow phosphorus. This form of intoxication is characterized by periostitis with suppuration, ulceration, necrosis, and severe deformity of the lower jaw. *See Phosphine.*

### *Special Diagnostic Test*

Roentgenographic examination of lower jaw to detect possible necrosis of mandible. *See Patty, 1958.*

### *Recommended Threshold Limit*

*Phosphorus (yellow)*, 0.1 milligram per cubic meter of air.

*Phosphoric acid*, 1 milligram per cubic meter of air.

*Phosphorus pentachloride*, 1 milligram per cubic meter of air.

*Phosphorus pentasulfide*, 1 milligram per cubic meter of air.

*Phosphorus trichloride (gas)*, 3 milligrams per cubic meter of air or 0.5 part per million parts of air by volume.

### *Potential Occupational Exposures*

#### *Phosphorus (white or yellow)*

Bronze alloy makers

Electroluminescent coating  
makers

Fertilizer makers

Fireworks makers

Incendiary makers

Metallic phosphide makers

Metal refiners

Munitions workers

Pesticide workers

Phosphoric acid makers

Phosphoric anhydride makers

Phosphorus workers

Rat poison workers

Red phosphorus makers

Semiconductor makers

Smoke bomb makers

#### *Phosphoric acid*

Activated carbon makers

Animal feed makers

Ceramic makers

Dental cement makers

Detergent makers

Drug makers

Electropolishers

Engravers

Fertilizer makers

Flavoring syrup makers

Foundry workers

Gelatin makers

Lithographers

Metal cleaners

Phosphate makers

Phosphoric acid workers

Photoengravers

Polish makers

Rubber latex makers

Rust inhibitor makers

Rustproofers

Soft drink makers

Sugar refiners

Water treaters

Wax makers

Yeast makers

*Phosphorus trichloride*

Agricultural chemical makers  
Chlorinated compound makers  
Dye makers  
Gasoline additive makers  
Phosphorus oxychloride makers

Phosphorus trichloride workers

Plasticizer makers  
Saccharin makers  
Surfactant makers

*Phosphorus pentachloride*

Acetylcellulose makers  
Agricultural chemical makers  
Chlorinated compound makers

Organic chemical synthesizers  
Phosphorus oxychloride makers  
Phosphorus pentachloride workers

*Phosphorus pentasulfide*

Agricultural chemical makers  
Flotation agent makers  
Insecticide makers  
Lubricating oil additive makers

Match makers  
Organic chemical synthesizers  
Phosphorus pentasulfide workers  
Rubber chemical makers

*Phosphorus oxychloride*

Agricultural chemical makers  
Chlorinated compound makers  
Drug makers  
Gasoline additive makers  
Hydraulic fluid makers

Organic chemical synthesizers  
Organic phosphate makers  
Phosphorus oxychloride workers  
Plasticizer makers

*References*

CALEY, J. P. AND KELLOCK, I. A.: Acute yellow phosphorus poisoning with recovery. *Lancet* 1: 539, 1955.

HEIMANN, H.: Chronic phosphorus poisoning. *J. Indust. Hyg. and Toxicol.* 28: 142, 1946.

PATTY, F. A. (EDITOR): *Industrial Hygiene and Toxicology*. 2nd ed. Vol. 1. Interscience Publishers, New York, 1958.

RUBITSKY, H. J. AND MYERSON, R. M.: Acute phosphorus poisoning. *Arch. Int. Med.* 83: 164, 1949.

(151) Phthalic Anhydride  
*phthalic acid anhydride*

*Harmful Effects*

*Local* Phthalic anhydride in pure state is not an irritant, but after contact with water, the caustic phthalic acid is formed. Local irritation may produce conjunctivitis and contact dermatitis. Contact dermatitis may also

be caused by allergic hypersensitivity. Irritation of upper respiratory tract is manifested by epistaxis, atrophy of the nasal mucous membranes, loss of sense of smell, and hoarseness.

*Route of Entry* Inhalation of fume or dust.

*Systemic* Bronchitis, emphysema, asthma, and urticaria may occur.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

Not established.

### Potential Occupational Exposures

Alcohol denaturant makers	Insecticide makers
Alizarin dye makers	Isophthalic acid makers
Alkyd resin makers	Methyl aniline purifiers
Anthranilic acid makers	Mylar plastic makers
Anthraquinone makers	Organic chemical synthesizers
Automobile finish makers	Phenolphthalein makers
Benzoic acid makers	Photographic film makers
Cellulose acetate plasticizer makers	Phthalamide makers
Dacron fiber makers	Phthalate ester makers
Diethylphthalate makers	Phthalein makers
Dimethylphthalate makers	Phthalic anhydride workers
Drug makers	Plasticizer makers
Dye makers	Repellent makers
Enamel makers	Resin makers
Eosin makers	Sulfathalidine makers
Erythrosin makers	Terephthalic acid makers
Herbicide makers	Vat dye makers
Indigo makers	Vinyl plasticizer makers

### Reference

MERLEVEDE, E. AND ELSKENS, J.: The toxicity of phthalic anhydride, maleic anhydride and the phthalates. *Arch. belges med. sociale, hyg., med. travail et med. legale* 15: 445, 1957. (Abst., *Bull. Hyg.* 33: 1151, 1958)

### (152) Picric Acid

*picronitric acid, trinitrophenol, nitroxanthic acid, carbazotic acid, phenol trinitrate*

### Harmful Effects

*Local* Contact dermatitis from either primary irritation or allergic hypersensitivity, yellow coloring of skin and hair, conjunctivitis, keratitis, and yellow vision.

*Routes of Entry* Percutaneous absorption from dust, inhalation of dust, and ingestion.

*Systemic* Headache, vertigo, convulsions, gastroenteritis, sometimes hepatitis and hemorrhagic nephritis.

### *Special Diagnostic Test*

Analysis of urine for picric acid. See Von Oettingen, 1958.

### *Recommended Threshold Limit*

0.1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Battery makers	Glass makers, colored
Copper etchers	Histology technicians
Disinfectant makers	Match makers
Drug makers	Picrate makers
Dye makers	Picric acid workers
Dyers	Shell fillers
Explosive makers	Tannery workers
Fireworks makers	Textile dyers
Forensic chemists	Textile printers

### *References*

VON OETTINGEN, W. F.: The aromatic amino and nitro compounds, their toxicity and potential dangers. A review of the literature. Pub. Health Bull. No. 271. U.S. Government Printing Office, Washington, D.C., 1941.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment.* 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (153) Platinum and Compounds

### *Harmful Effects*

*Local* Metallic platinum is nontoxic. Platinum salts may act as skin irritants and skin sensitizers to produce contact dermatitis; irritation of nose and throat has been reported to follow exposure to these salts.

*Route of Entry* Inhalation of dust or mist.

*Systemic* Sodium chloroplatinate (platinic sodium chloride) has been implicated as the etiologic agent responsible for syndrome called *platinosis*. This condition consists of mild chemical irritation and chronic inflammation of entire respiratory tract associated, in some cases, with allergic manifestations affecting skin. In more severe cases, symptoms of typical bronchial asthma may become evident.

### *Special Diagnostic Test*

None.

*Recommended Threshold Limit*

(Soluble salts) 0.002 milligram per cubic meter of air.

*Potential Occupational Exposures*

Alloy makers	Jewelry makers
Ceramic workers	Laboratory ware makers
Dental alloy makers	Laboratory workers, chemical
Drug makers	Microscopists
Electronic equipment makers	Mirror makers
Electroplaters	Platinum workers
Glass makers	Spark plug makers
Ink makers, indelible	Zinc etchers

*Reference*

ROBERTS, A. E.: Platinosis. A five-year study of the effects on employees in a platinum laboratory and refinery. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 4: 549, 1951.

## (154) Propyl Acetate

*Harmful Effects*

*Local* May be irritating to skin and mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of vapor.

*Systemic* No industrial poisonings have been reported. In high concentrations vapors may depress central nervous system.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

200 parts per million parts of air by volume or 840 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Flavoring makers	Perfume makers
Lacquer makers	Propyl acetate workers
Nitrocellulose workers	Resin makers
Organic chemical synthesizers	Varnish makers

## (155) n-Propyl Alcohol

*1-propanol, propylic alcohol*

*Harmful Effects*

*Local* Vapors are mildly irritating to conjunctiva and mucous membranes of upper respiratory tract.

*Route of Entry* Inhalation of vapor.

*Systemic* No cases of industrial poisoning have been recorded. Vapor can produce mild central nervous system depression.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

Not established.

### *Potential Occupational Exposures*

Brake fluid makers	Propionaldehyde makers
Cosmetic makers	Propionic acid makers
Disinfectant makers	n-Propyl acetate makers
Dope processors	n-Propyl alcohol workers
Drug makers	n-Propylated urea makers
Ink makers, printing	Resin makers
Lacquer makers	Soap makers
Metal degreasers	Solvent makers
Nitrocellulose workers	Vegetable oil processors
Organic chemical synthesizers	Wax makers
Polish makers	Window cleaning fluid makers

### *Reference*

HENSON, E. V.: The toxicology of some aliphatic alcohols; part 1. *J. Occup. Med.* 2: 442, 1960.

### (156) Propylene Dichloride

*1,2-dichloropropane, propylene chloride*

### *Harmful Effects*

*Local* Repeated or prolonged contact with liquid can produce a dry, scaly, fissured dermatitis. May be irritating to eyes and other mucous membranes.

*Route of Entry* Inhalation of vapor.

*Systemic* Produces marked narcosis. May cause fatty degeneration of liver, kidneys and heart.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

75 parts per million parts of air by volume or 350 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Cellulose plastic makers	Organic chemical synthesizers
Dry cleaners	Propylene dichloride workers
Dry cleaning fluid makers	Rubber makers
Fat processors	Scouring compound makers
Fumigant workers	Solvent workers
Gum processors	Stain removers
Metal degreasers	Wax makers
Oil processors	

*Reference*

HEPPEL, L. A.; NEAL, P. A.; HIGHMAN, B., AND PORTERFIELD, V. T.: Toxicology of 1,2-dichloropropane (propylene dichloride). I, Studies on effects of daily inhalations. *J. Indust. Hyg. & Toxicol.* 28: 1, 1946.

(157) Pyrethrum. *See* Pesticides Section

(158) Pyridine

*Harmful Effects*

*Local* Liquid and vapor are irritating to eyes, nose, and throat. Repeated or prolonged contact with liquid can produce a dry, scaly, fissured dermatitis. Photosensitization dermatitis can occur.

*Routes of Entry* Inhalation of vapor and percutaneous absorption of liquid.

*Systemic* Acute exposure produces flushing of the face and narcotic effects of nausea, vomiting, dizziness, and drowsiness. Effects of chronic exposure include headache, nervousness, insomnia, and other neurologic disturbances, but these have also been reported following acute and subacute exposures.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

5 parts per million parts of air by volume or 15 milligrams per cubic meter of air.

*Potential Occupational Exposures*

Acrylonitrile makers	Dyers
Adhesive workers	Explosive workers
Alcohol denaturant makers	Furniture polishers
Anhydrous salt processors	Gas mantle makers
Denatured alcohol makers	Gilders
Drug makers	Lacquerers

Lacquer makers	Rubber makers
Latex workers	Rust inhibitor workers
Niacin makers	Solvent workers
Organic chemical synthesizers	Styrene makers
Paint makers	Sulfapyridine makers
Pencil makers	Textile dyers
Pyridine workers	Vitamin makers
Rubber chemical makers	Waterproofing makers

### Reference

BALDI, G.: Occupational pathology from pyridine. *Med. Lavoro* 44: 244, 1953.

### (159) Quinone

*benzoquinone, chinone*

#### Harmful Effects

*Local* Quinone in solid form, solutions, or vapor phase can produce contact dermatitis from primary irritation. Condensation of quinone vapors on eyes produces conjunctivitis, lacrimation, photophobia, corneal stains, ulcerations, and opacities.

*Route of Entry* Inhalation of vapor.

*Systemic* None has been reported from inhalation or ingestion in humans. In animal studies, ingestion or subcutaneous injection of quinone has produced convulsions, respiratory difficulties, hypotension, and asphyxia.

#### Special Diagnostic Test

Analysis of urine for hydroquinone. See Von Oettingen, 1958.

#### Recommended Threshold Limit

0.1 part per million parts of air by volume or 0.4 milligram per cubic meter of air.

#### Potential Occupational Exposures

Dye makers	Photographic film developers
Gelatin makers	Protein fiber makers
Hydrogen peroxide makers	Quinone workers
Hydroquinone makers	Tanners
Laboratory workers, chemical	

#### References

STERNER, J. H.; OGLESBY, F. L., AND ANDERSON, B.: Quinone vapors and their harmful effects. I, Corneal and conjunctival injury. *J. Indust. Hyg. & Toxicol.* 29: 60, 1947.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

(160) Radon. *See* Uranium and Compounds

(161) Selenium and Compounds

### *Harmful Effects*

*Local* Compounds of selenium are potent skin and mucous membrane irritants. Selenium, selenium oxide, selenic acid, selenic acid anhydride, selenium sulfide, potassium selenite, sodium selenite, selenium bromide, hydrogen selenide, methyl selenide, selenious acid, and selenium oxychloride produce degrees of dermatitis varying from erythema to severe burns with vesiculation. These compounds may produce nose, throat, and eye irritation with sneezing, nasal congestion, anosmia, coughing, perspiration, lacrimation, palpebral edema, and conjunctivitis.

*Route of Entry* Inhalation of dust or vapor; percutaneous absorption of liquid.

*Systemic* Acute systemic poisoning in industry is rare since these compounds are sufficiently irritant to compel the workers to leave an area of high exposure. Nervousness, dizziness, weakness, nausea, vomiting, diarrhea, abdominal pain, somnolence, dyspnea, garlic-like odor of the breath, convulsions, and death may result from severe exposure. Pulmonary edema may be produced by the more volatile selenium compounds, such as selenium oxide and hydrogen selenide. Percutaneous absorption of a selenium sulfide compound used in certain shampoo mixtures has been reported to produce loss of hair in a few persons and, in one case, symptoms compatible with systemic toxicity.

Chronic exposure is characterized by nausea, vomiting, possible liver damage, nervousness, tremor, metallic taste, garlic-like odor of the breath, pallor, dizziness, and fatigue.

### *Special Diagnostic Test*

Analysis of urine for selenium. *See* Elkins, 1959.

### *Recommended Threshold Limits*

*Hydrogen selenide*, 0.05 part per million parts of air by volume or 0.2 milligram per cubic meter of air.

*Selenium compounds (as Se)*, 0.1 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Arc light electrode makers	Copper smelters
Bacteriologists	Drug makers
Barbers	Electric rectifier makers
Brass founders	Electroplaters
Cement workers	Glass makers
Ceramic makers	Hairdressers

Ink makers	Plastic workers
Lead smelters	Pyrite roasters
Leather workers	Rubber makers
Lime workers	Seed germination testers
Lubricating oil makers	Selenium workers
Microscopists	Semiconductor makers
Organic chemical synthesizers	Shampoo makers
Paint makers	Stainless steel makers
Paper makers	Stenching agent makers
Pesticide makers	Sulfuric acid makers
Phosphor makers	Textile workers
Photoelectric cell makers	Xerographic plate makers
Photographic chemical makers	Zinc smelters
Pigment makers	

### References

BUCHAN, R. F.: Industrial selenosis. *Occup. Med.* 3: 439, 1947.

CERWENKA, E. A., JR. AND COOPER, W. CHARLES: Toxicology of selenium and tellurium, and their compounds. *Arch. Environ. Health* 3: 189, 1961.

CLINTON, M., JR.: Selenium fume exposure. *J. Indust. Hyg. & Toxicol.* 29: 225, 1947.

ELKINS, H. B.: *The Chemistry of Industrial Toxicology*. 2nd ed. John Wiley and Sons, New York, 1959.

GLOVER, J. R.: Some medical problems concerning selenium in industry. *Trans. Assoc. Indust. Med. Officers* 4: 94, 1954.

GROVER, R. W.: Diffuse hair loss associated with selenium (Selsun) sulfide shampoo. *J. Am. Med. Assoc.* 160: 1397, 1956.

RANSONE, J. W.; SCOTT, N. M., JR., AND KNOBLOCK, E. C.: Selenium sulfide intoxication. *New Eng. J. Med.* 264: 384, 1961.

### (162) Silver and Compounds

#### Harmful Effects

*Local* Localized industrial argyria (argyriism) is caused by implantation of silver particles in skin and is manifested as small bluish-black spots, usually on hands and forearms. Silver nitrate is irritating to skin and mucous membranes and can temporarily discolor skin.

*Routes of Entry* Inhalation of dust; ingestion of solutions or dust.

*Systemic* Industrial argyria from ingestion of silver compounds has been reported, but is no longer seen. It resembled the bluish-gray discoloration of eyes and skin seen in generalized argyria from therapeutic ingestion or injection of silver salts. Depth of color in argyria is greater in those areas exposed to light.

When silver or its salts are inhaled in industrial exposures, much of the silver is deposited in elastic tissue of lungs (pulmonary argyria), but eventually the bluish-gray discoloration appears in eyes and skin. Bron-

chitis and emphysema have been described in workers with pulmonary argyria, but a cause and effect relationship has not been demonstrated. Except for its cosmetic disfigurement, argyria is generally considered to be benign.

### *Special Diagnostic Tests*

Analysis of blood for excessive amounts of silver is helpful only during exposure. Examination of skin with ultraviolet lamp and of cornea with slit lamp. See Blumberg and Carey, 1934; Harker and Hunter, 1935, and Holden, 1950.

### *Recommended Threshold Limit*

(Tentative) 0.05 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Algicide makers	Ink makers, indelible
Alloy makers	Ivory etchers
Artificial rain makers	Jewelry makers
Bactericide makers	Lead refiners
Battery makers	Metal inlayers
Bearing metal makers	Mirror makers
Brazing rod makers	Optical workers
Ceramic makers	Organic chemical synthesizers
Chemical equipment makers	Paint makers
Copper refiners	Photographic chemical makers
Cutlery makers	Photographic film makers
Dental alloy makers	Silver bromide makers
Drug makers	Silver engravers
Electric conductor makers	Silver finishers
Electric equipment makers	Silver nitrate makers
Electronic workers	Silver platers
Electrotype makers	Silver polishers
Food product equipment makers	Silver reclaimers
Gas mask makers	Silversmiths
Glass makers	Silver workers
Glass polish makers	Solder workers, hard
Gold refiners	Water treaters
Hair dye makers	

### *References*

BLUMBERG, H. AND CAREY, T. N.: Argyremia; detection of unsuspected and obscure argyria by the spectrographic demonstration of high blood silver. *J. Am. Med. Assoc.* 103: 1521, 1934.

BROWNING, E.: *Toxicity of Industrial Metals*. Butterworths, London, 1961.

HARKER, J. M. AND HUNTER, D.: Occupational argyria. *Brit. J. Dermat.* 47: 441, 1935.

Examination with slit lamp.

HEIMANN, H.: Toxicity of metallic silver. *Indust. Bull.* (N.Y. State Dept. Labor) 22: 81, 1943.

HILL, W. R. AND PILLSBURY, D. M.: *Argyria*. Williams & Wilkins Co., Baltimore, 1939.

HOLDEN, R. F., JR.: Observations in argyria. *J. Lab. & Clin. Med.* 36: 837, 1950.

MONTANDON, M. A.: Argyrose des voies respiratoires. *Arch. Mal. Prof.* 20: 419, 1959.

### (163) Sodium and Potassium Hydroxides

Aqueous solution of sodium hydroxide (caustic soda or caustic alkali) or potassium hydroxide (caustic potash or caustic alkali) is known as lye; the sodium hydroxide solution is also referred to as soda lye. Sodium hydroxide added to calcium oxide produces soda lime (See Calcium Oxide). Water added to calcium oxide (lime or quicklime) produces calcium hydroxide or slaked lime. Washing soda (soda ash or sal soda) is sodium carbonate combined with 10 molecules of water. Baking soda is sodium bicarbonate. Chloride of lime (which see) is a mixture of calcium chloride, calcium hypochlorite and calcium hydroxide.

#### *Harmful Effects*

*Local* Both compounds exert an extremely corrosive action on skin, eyes and mucous membranes.

*Route of Entry* Inhalation of dust or mist.

*Systemic* Systemic effects are due entirely to local tissue injury. Extreme pulmonary irritation may result from inhalation of dust or mist.

#### *Special Diagnostic Test*

None.

#### *Recommended Threshold Limit*

*Sodium hydroxide*, 2 milligrams per cubic meter of air.

*Potassium hydroxide*, not established.

#### *Potential Occupational Exposures*

Bleachers	Lithographers
Bleach makers	Match makers
Bronzers	Mercerizers
Degreasers	Oxalic acid makers
Detergent makers	Paint removers
Electroplaters	Paper makers
Enamelers	Perfume makers
Engravers	Petroleum refinery workers
Etchers	Photoengravers
Furniture polishers	Potassium hydroxide workers
Housekeepers	Printers
Laboratory workers, chemical	Printing ink makers
Laundry workers	Pulp makers

Rayon makers  
Rubber reclaimers  
Soap makers  
Sodium hydroxide workers

Textile bleachers  
Varnish removers  
Vegetable oil refiners

### (164) Styrene

*cinnamene, cinnamenol, cinnamol, phenethylene, phenylethylene, styrene monomer, styrol, styrolene, vinylbenzene*

#### *Harmful Effects*

*Local* Styrene and its vapor are irritating to eyes, nose, and throat. Styrene liquid is a low-grade cutaneous irritant, and repeated contact with skin will produce a dry, scaly, and fissured dermatitis.

*Route of Entry* Inhalation of vapor.

*Systemic* Styrene sickness with symptoms of headache, fatigue, nausea, vomiting, anorexia, dizziness, and drowsiness has occurred from narcotic effect of vapor. Respiratory tract irritation by high vapor concentrations is manifested by cough. No chronic systemic effects have been reported.

#### *Special Diagnostic Test*

Determination of urinary hippuric acid excretion has been suggested; however, it is not specific. See Gerarde, 1960.

#### *Recommended Threshold Limit*

100 parts per million parts of air by volume or 420 milligrams per cubic meter of air.

#### *Potential Occupational Exposures*

Adhesive makers  
Boat makers  
Emulsifier agent makers  
Insulator makers  
Organic chemical synthesizers  
Petroleum refinery workers  
Plastic luggage makers  
Polyester resin laminators

Polystyrene makers  
Potting compound workers  
Protective coating makers  
Rubber makers  
Sports car body makers  
Styrene workers  
Swimming pool makers  
Varnish makers

#### *References*

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GERARDE, H. W.: *Toxicology and Biochemistry of Aromatic Hydrocarbons*. Elsevier Publishing Co., Amsterdam, and Princeton, N.J., 1960.

ROGERS, J. C. AND HOOPER, C. C.: An industrial hygiene problem in the plastics field. *Modern Sanit.* 8: 19, (Aug.) 1956.

WILSON, R. H.: Health hazards encountered in the manufacture of synthetic rubber. *J. Am. Med. Assoc.* 124: 701, 1944.

### (165) Sulfur Dioxide

*sulfurous anhydride, sulfurous oxide*

#### *Harmful Effects*

**Local** Gaseous sulfur dioxide is irritant to conjunctiva and mucous membranes of upper respiratory tract. High exposure may produce laryngeal edema and death from asphyxiation. Liquid sulfur dioxide is skin irritant. Corneal injury with blindness has resulted from liquid splashes into eyes.

**Route of Entry** Inhalation of gas.

**Systemic** Severe acute symptoms are unusual since gas is sufficiently irritant to compel the workers to flee. Inhalation of high concentrations may produce bronchitis, pneumonitis, pulmonary edema, and death.

Studies of chronic sulfur dioxide exposure in humans have indicated no appreciable danger to health. Nasopharyngitis, fatigue, altered sense of taste and smell, and dyspnea on exertion have been said to result from long continued low exposures.

#### *Special Diagnostic Test*

None.

#### *Recommended Threshold Limit*

5 parts per million parts of air by volume or 13 milligrams per cubic meter of air.

#### *Potential Occupational Exposures*

Beet sugar bleachers	Glass makers
Boiler water treaters	Glue bleachers
Brewery workers	Grain bleachers
Diesel engine operators	Ice makers
Diesel engine repairmen	Meat preservers
Disinfectant makers	Oil bleachers
Disinfectors	Oil processors
Firemen	Ore smelter workers
Flour bleachers	Organic sulfonate makers
Food bleachers	Paper makers
Foundry workers	Petroleum refinery workers
Fruit bleachers	Preservative makers
Fumigant makers	Protein makers, food
Fumigators	Protein makers, industrial
Furnace operators	Refrigeration workers
Gelatin bleachers	Straw bleachers

Sugar refiners	Thermometer makers, vapor pressure
Sulfite makers	Thionyl chloride makers
Sulfur dioxide workers	Wicker ware bleachers
Sulfuric acid makers	Wine makers
Sulfuryl chloride makers	Wood pulp bleachers
Tannery workers	Wool bleachers
Textile bleachers	

### References

ANDERSON, A.: Possible long term effects of exposure to sulfur dioxide. *Brit. J. Indust. Med.* 7: 82, 1950.

KEHOE, R. A.; MACHEL, W. F.; KITZMILLER, K., AND LEBLANC, T. J.: On the effects of prolonged exposure to sulfur dioxide. *J. Indust. Hyg.* 14: 159, 1932.

### (166) Sulfuric Acid

*oil of vitriol, spirit of vitriol, hydrogen sulfate*

#### Harmful Effects

**Local** Sulfuric acid is irritant to conjunctiva and mucous membranes of the upper respiratory tract. The acid may also produce erosion of teeth, usually the incisors. Liquid may produce severe burns and ulceration of skin.

**Route of Entry** Inhalation of vapor.

**Systemic** Systemic effects are not well recognized. Human experimental studies have revealed that rapid shallow respiration may occur following exposure to low concentrations of sulfuric acid mist below the taste-odor-irritation threshold. Pulmonary fibrosis, bronchiectasis, and emphysema have been reported from acute exposure to fuming sulfuric acid and sulfuric acid mist.

#### Special Diagnostic Test

None.

#### Recommended Threshold Limit

1 milligram per cubic meter of air.

#### Potential Occupational Exposures

Aluminum sulfate makers	Explosive makers
Ammonium sulfate makers	Fertilizer makers
Battery makers, storage	Food processors
Cellulose workers	Fur processors
Copper sulfate makers	Galvanizers
Detergent makers	Glue makers
Drug makers	Jewelers
Dye makers	Laboratory workers, chemical
Electroplaters	Metal cleaners

Paint makers	Pigment makers
Paper makers	Rayon workers
Petrochemical workers	Rubber workers
Petroleum refinery workers	Steel workers
Phenol makers	Sulfuric acid workers
Phosphate workers	Textile workers
Phosphoric acid makers	

### References

AMDUR, M. O.; SILVERMAN, L., AND DRINKER, P.: Inhalation of sulfuric acid mist by human subjects. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 305, 1952.

GOLDMAN, A. AND HILL, W. T.: Chronic bronchopulmonary disease due to inhalation of sulfuric acid fumes. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 8: 205, 1953.

MALCOLM, D. AND PAUL, E.: Erosion of the teeth due to sulfuric acid in the battery industry. *Brit. J. Indust. Med.* 18: 63, 1961.

### (167) Sulfur Monochloride *sulfur chloride, sulfur subchloride*

#### Harmful Effects

**Local** Sulfur monochloride liquid may cause severe skin burns and its vapors are very irritating to eyes and mucous membranes of nose, throat, and trachea.

**Route of Entry** Inhalation of vapor.

**Systemic** Although this compound is almost certainly capable of producing severe lung irritation, very few serious cases of industrial exposure have been reported. This is probably because the pronounced irritant effects of sulfur monochloride serve as an immediate warning signal when concentration of the gas approaches a hazardous level.

#### Special Diagnostic Test

None.

#### Recommended Threshold Limit

1 part per million parts of air by volume or 6 milligrams per cubic meter of air.

#### Potential Occupational Exposures

Carbon tetrachloride makers	Sugar juice purifiers
Drug makers	Sulfur dye makers
Dyers	Sulfur monochloride workers
Gold extractors	Textile dyers
Insecticide makers	Textile finishers
Organic chemical synthesizers	Vegetable oil processors
Rubber cement makers	Vulcanized oil makers
Rubber makers	Vulcanizers
Rubber substitute makers	Wood hardeners

## (168) Tellurium

*aurum paradoxum, metallum problematum**Harmful Effects**Local* No local effects have been reported.*Routes of Entry* Inhalation of dust or fume; percutaneous absorption from dust.*Systemic* Exposure to dust or fume may produce a persistent garlic-like odor of breath (due to methyl telluride), suppression of perspiration, metallic taste, nausea, anorexia, and somnolence.*Special Diagnostic Test*

Analysis of tellurium in urine and feces. See Steinberg et al., 1942.

*Recommended Threshold Limit*

0.1 milligram per cubic meter of air.

*Potential Occupational Exposures*

Alloy makers	Porcelain makers
Ceramic makers	Rubber makers
Copper alloy makers	Semiconductor makers
Copper refinery workers	Silverware makers
Electronic workers	Stainless steel makers
Enamel makers	Tellurium lead alloy makers
Foundry workers	Tellurium workers
Glass makers	Thermoelectric device makers
Iron makers	Vulcanizers
Lead refinery workers	

*References*

CERWENKA, E. A., JR., AND COOPER, W. CHARLES: Toxicology of selenium and tellurium, and their compounds. *Arch. Environ. Health* 3: 189, 1961.

GLASS, P. K.: Toxicity of tellurium. United States Atomic Energy Commission AECU-374. The Commission, Washington, D.C., 1948.

QUERIES AND MINOR NOTES: Effect of tellurium on health. *J. Am. Med. Assoc.* 155: 1548, 1954.

STEINBERG, H. H.; MASSARI, S. C.; MINER, A. C., AND RINK, R.: Industrial exposure to tellurium; atmospheric studies and clinical evaluation. *J. Indust. Hyg. & Toxicol.* 24: 183, 1942.

## (169) Tetrachloroethane

*sym.-tetrachloroethane, acetylene tetrachloride**Harmful Effects**Local* Repeated or prolonged contact with this low grade primary irritant can produce a scaly and fissured dermatitis.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Most toxic of all of chlorinated hydrocarbons. Early effects are caused by its narcotic action. Later, liver damage may be severe resulting in acute yellow atrophy of this organ. Fatty degeneration of kidneys and myocardium may be produced.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

5 parts per million parts of air by volume or 35 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Biologists	Organic chemical synthesizers
Cellulose acetate workers	Paint makers
Denatured alcohol workers	Paint remover workers
Ethyl chloride makers	Phosphorus processors
Ethylene dichloride makers	Photographic film makers
Fat processors	Resin makers
Fumigant makers	Rubber makers
Fumigators	Rust remover workers
Gasket makers	Soil treaters
Herbicide workers	Solvent workers
Insecticide workers	Sulfur processors
Lacquer workers	Tetrachloroethane workers
Metal cleaners	Trichloroethylene makers
Metal degreasers	Varnish workers
Mineralogists	Waxers
Oil processors	Wax makers

### *Reference*

VON OETTINGEN, W. F.: The halogenated aliphatic, olefinic, cyclic, aromatic, and aliphatic-aromatic hydrocarbons including the halogenated insecticides; their toxicity and potential dangers. Pub. Health Service Pub. No. 414. U.S. Government Printing Office, Washington, D.C., 1955.

## (170) Tetraethyl Lead

*TEL*

### *Harmful Effects*

*Local* Liquid may penetrate the skin without producing appreciable local injury.

*Routes of Entry* Percutaneous absorption of liquid, inhalation of vapor.

*Systemic* Signs and symptoms resulting from tetraethyl lead intoxication differ from those of inorganic lead poisoning. Central nervous system

effects predominate. Symptoms are usually delayed for a few hours to several days following exposure. Insomnia, headaches, nightmares; nervousness, irritability, and vague gastrointestinal symptoms may appear early. If the illness is severe, patients frequently experience episodes of maniacal behavior. Fatigue, weakness, weight loss, muscular pains, tremors, slow pulse, and low blood pressure also characterize the acute illness. Prognosis will depend upon severity of exposure. Many deaths have been recorded in literature. Those patients who recover generally exhibit no sequelae.

### *Special Diagnostic Tests*

Analysis of blood and urine for lead. See Fleming et al., 1960.

### *Recommended Threshold Limit*

Not established.

### *Potential Occupational Exposures*

Storage tank cleaners	Tetraethyl lead mixers
Tetraethyl lead blenders	Tetraethyl lead workers
Tetraethyl lead makers	

### *References*

ADVISORY COMMITTEE ON TETRAETHYL LEAD TO SURGEON GENERAL OF PUBLIC HEALTH SERVICE: Public health aspects of increasing tetraethyl lead content in motor fuel. Pub. Health Service Pub. No. 712. U. S. Government Printing Office, Washington, D.C., 1959.

CREMER, J. E. AND CALLAWAY, S.: Further studies on the toxicity of some tetra and trialkyl lead compounds. *Brit. J. Indust. Med.* 18: 277, 1961.

FLEMING, A. J.; D'ALONZO, C. A., AND ZAPP, J. A. (EDITORS): *Modern Occupational Medicine*. 2nd ed. Lea & Febiger, Philadelphia, 1960.

KEHOE, R. A.: Tetraethyl lead, the disposition of an international chemical hazard. In *Proceedings Thirteenth International Congress on Occupational Health*, New York, July 25-29, 1960. U.S. Executive Committee of the Congress (L. Wade, M.D., chairman), New York, 1961.

VARIOUS AUTHORS: *Lead Symposium*, February 25-27, 1963. University of Cincinnati, Cincinnati, Ohio, 1963.

## (171) Tetramethyl Lead

### *TML*

### *Harmful Effects*

*Local* Same as tetraethyl lead, which see.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid. TML is more volatile than TEL and therefore may present more of an inhalation hazard.

*Systemic* Similar to tetraethyl lead as indicated by animal experimentation, but is less readily absorbed through intact skin.

*Special Diagnostic Tests*

Same as tetraethyl lead.

*Recommended Threshold Limit*

Not established.

*Potential Occupational Exposures*

Storage tank cleaners	Tetramethyl lead mixers
Tetramethyl lead blenders	Tetramethyl lead workers
Tetramethyl lead makers	

*References*

CREMER, J. E. AND CALLAWAY, S.: Further studies on the toxicity of some tetra and trialkyl lead compounds. *Brit. J. Indust. Med.* 18: 277, 1961.

DETREVILLE, R. T. P.; WHEELER, H. W., AND STERLING, T.: Occupational exposure to organic lead compounds. The relative degree of hazard in occupational exposure to air-borne tetraethyl lead and tetramethyl lead. *Arch. Environ. Health* 5: 532, 1962.

KEHOE, R. A.; CHOLAK, J.; SPENCE, J. A., AND HANCOCK, W.: Potential hazard of exposure to lead. 1, Handling and use of gasoline containing tetramethyl lead. *Arch. Environ. Health* 6: 239, 1963.

KEHOE, R. A.; CHOLAK, J.; MCILHINNEY, J. G.; LOFQUIST, G. A., AND STERLING, T. D.: Potential hazard of exposure to lead. 2, Further investigations in the preparation, handling, and use of gasoline containing tetramethyl lead. *Arch. Environ. Health* 6: 255, 1963.

VARIOUS AUTHORS: *Lead Symposium*, February 25-27, 1963. University of Cincinnati, Cincinnati, Ohio, 1963.

### (172) Tetramethylthiuram Disulfide

*thiram, bis-(dimethylthiocarbamyl) disulfide, thirad, thiuram, TMTD*

*Harmful Effects*

*Local* Irritation of skin and mucous membranes of eyes and upper respiratory tract. Allergic contact dermatitis.

*Route of Entry* Inhalation of dust.

*Systemic* No systemic poisoning has been reported in United States. On basis of animal experiments and reports in foreign literature, bronchitis, liver, and kidney damage may be expected from exposure to high concentrations. Intolerance to alcohol has been observed, manifested by flushing of face, palpitation, rapid pulse, dizziness, and hypotension.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

5 milligrams per cubic meter of air.

## Potential Occupational Exposures

Bacteriostat makers, soap	Rubber makers, heat resistant
Dock workers	Seed disinfectors
Fungicide workers	Soap makers
Insecticide workers	Tetramethylthiuram disulfide
Japanese beetle repellent makers	workers
Lubricating oil blenders	Vulcanizers
Rat repellent makers	

## References

FINULLI, M. AND MAGISTRETTI, M.: Antabuse-like intoxication in workmen employed in the manufacture of the synthetic agricultural chemical TMTD (tetramethylthiuram disulfide). *Med. Lavoro* 52: 132, 1961.

LAVARINO, A. AND MASOERO, A.: Acute poisoning by tetramethylthiuram disulfide. *Rass. med. ind.* 24: 458, 1955. (*Indust. Hyg. Digest*, Abst. No. 883, July 1956)

SCHULZ, K. H. AND HERRMANN, W. P.: Tetramethylthiuram disulfide, a thiourea derivative, as an agent provocative of dermatitis in dock laborers. *Berufsdermatosen* 6: 130, 1958. (*Indust. Hyg. Digest*, Abst. No. 162, February 1958)

## (173) Tetryl

*trinitrophenylmethylnitramine, nitramine, tetranitromethylaniline, pyrenite, picrylmethylnitramine, picrylnitromethylamine*

## Harmful Effects

**Local** Tetryl is a potent sensitizer, and allergic contact dermatitis is common. Contact may stain skin and hair yellow or orange; workers with such stains have been referred to as canaries. Tetryl dust is sometimes irritating to eyes and nose, causing conjunctivitis, sneezing, and epistaxis.

**Route of Entry** Inhalation of dust.

**Systemic** Cough is a common symptom among workers initially exposed to large amounts of dust, but chest roentgenograms reveal no pulmonary disease. Systemic intoxication is practically never encountered. In the few cases of liver damage that have been reported, exposure was massive. Tetryl workers are frequently exposed to trinitrotoluene and other explosives, making it difficult to establish the specific agent producing the systemic symptoms.

## Special Diagnostic Test

Webster's reagent, a dilute solution of sodium hydroxide in ethyl alcohol, is discolored dark brown by tetryl on skin. See Norwood, 1943.

## Recommended Threshold Limit

1.5 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Ammunition makers  
Detonator makers  
Explosive workers

Indicator makers, chemical  
Tetryl workers

### *References*

BERGMAN, B. B.: Tetryl toxicity; a summary of ten years' experience. *A.M.A. Arch Indust. Hyg. & Occup. Med.* 5: 10, 1952.

HARDY, H. L. AND MALOOF, C. C.: Evidence of systemic effect of tetryl; with summary of available literature. *Arch. Indust. Hyg. & Occup. Med.* 1: 454, 1950.

NORWOOD, W. D.: Trinitrotoluene (TNT); its effective removal from the skin by a special liquid soap. *Indust. Med.* 12: 206, 1943.

## (174) Thallium and Compounds

### *Harmful Effects*

*Local* Some thallium salts may produce skin irritation.

*Routes of Entry* Inhalation of dust and fume. Ingestion and percutaneous absorption of dust.

*Systemic* Thallium may act as a *cumulative poison*; that is, repeated small doses which would individually produce little or no effect may be stored in body until a harmful or even lethal dose accumulates. Acute effects include severe gastroenteritis, abdominal pain, and collapse. Subacute or chronic effects include nausea, vomiting, leg and abdominal cramping, paresthesia of lower limbs, irritability, anorexia, stomatitis, dry scaly skin, metallic taste, garlic-like foul breath, visual disturbances, convulsions, delayed loss of hair, and kidney damage.

### *Special Diagnostic Test*

Analysis of urine or tissues for thallium. See Winn et al., 1952, and Jacobs, 1962.

### *Recommended Threshold Limit*

(Soluble compounds) 0.1 milligram per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Alloy makers  
Artificial diamond makers  
Carbon disulfide testers  
Chlorinated compound makers  
Depilatory makers  
Drug makers  
Dye makers  
Fireworks makers

Flotation workers  
Gem makers  
Glass makers, high refractive index  
Incandescent lamp makers  
Indicator makers, chemical  
Infrared instrument makers  
Insecticide workers

Match makers  
Optical glass makers  
Ore upgraders  
Organic chemical synthesizers  
Ozone testers

Photoelectric cell makers  
Rodenticide workers  
Textile workers  
Thallium workers

## References

JACOBS, M. B.: The determination of thallium in urine. *Am. Indust. Hyg. Assoc. J.* 23: 411, 1962.

RICHESON, E. M.: Industrial thallium intoxication. *Indust. Med. & Surg.* 27: 607, 1958.

TRUHAUT, R.: The toxicology of thallium. *J. Occup. Med.* 2: 334, 1960.

WINN, G. S.; GODFREY, E. L., AND NELSON, K. W.: Polarographic procedure for urinary thallium. *A.M.A. Arch. Indust. Hyg. & Occup. Med.* 6: 14, 1952.

## (175) Thorium and Compounds

### Harmful Effects

*Local* Thorium nitrate may produce a primary contact dermatitis.

*Routes of Entry* Ingestion of liquid; inhalation of dust or gas.

*Systemic* Thorium compounds have not been reported as causing systemic poisoning in industry.

Mesothorium (radium-228), a radioactive decay product of thorium, produced malignant tumors in radium dial painters.

Thorotrust, a thorium dioxide suspension used formerly in radiographic contrast studies, has been reported to cause malignancy and hematopoietic changes several years following injection; however, a cause and effect relationship has not been established.

### Special Diagnostic Tests

Analysis of blood and urine for thorium, breath for thoron, or feces for thorium X. See Fairhall, 1957, and Von Oettingen, 1958.

### Recommended Threshold Limit

Thorium-232,  $10^{-11}$  microcurie per cubic centimeter of air using *total body* as organ of reference. See National Committee on Radiation Protection, 1959, p. 83.

### Potential Occupational Exposures

Alloy makers, magnesium  
Ceramic makers  
Crucible makers  
Gas mantle makers  
Glass makers  
Incandescent lamp makers  
Luminous pigment workers

Metal refiners  
Nuclear reactor workers  
Organic chemical synthesizers  
Sunlamp makers  
Thorium workers  
Vacuum tube makers

## References

ALBERT, R.; KLEVIN, P.; FRESCO, J.; HARLEY, J.; HARRIS, W., AND EISENBUD, M.: Industrial hygiene and medical survey of a thorium refinery. *A.M.A. Arch. Indust. Health* 11: 234, 1955.

BAKER, W. H.; BULKLEY, J. B.; DUDLEY, R. A.; EVANS, R. D.; M'CLUSKEY, H. B.; REEVES, J. D., JR.; RYDER, R. H.; SALTER, L. P., AND SHANAHAN, M. M.: Observations on the late effects of internally deposited mixtures of mesothorium and radium in twelve dial painters. *New Eng. J. Med.* 265: 1023, 1961.

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NATIONAL COMMITTEE ON RADIATION PROTECTION: Maximum permissible body burdens and maximum permissible concentrations of radionuclides in air and in water for occupational exposure. National Bureau of Standards Handbook 69. Issued June 5, 1959. U.S. Government Printing Office, Washington, D.C., 1959.

ROBERTS, J. C. AND CARLSON, K. E.: Hepatic duct carcinoma seventeen years after injection of thorium dioxide. *A.M.A. Arch. Path.* 62: 1, 1956.

VON OETTINGEN, W. F.: *Poisoning, a Guide to Clinical Diagnosis and Treatment*. 2nd ed. W. B. Saunders Co., Philadelphia, 1958.

## (176) Tin and Compounds

### *stannum*

#### *Harmful Effects*

*Local* Inorganic salt, tin tetrachloride, may act as skin and mucous membrane irritant. Certain organo-tins, especially of the tri-butyl series are potent skin irritants.

*Routes of Entry* Inhalation of dust. Ingestion or percutaneous absorption of organo-tins.

*Systemic* Prolonged inhalation of small amounts of tin oxide dust may result in production of pseudo-nodulation in lung which may be easily seen on chest roentgenogram. This condition is referred to as *stannosis* and is considered to be nonprogressive and nondisabling. Inorganic tin compounds are relatively nontoxic and are not generally thought of as important industrial hazards. Organo-tin compounds have been reported, upon ingestion, to cause acute cerebral edema often resulting in death. These compounds, it is thought, may be absorbed through intact skin.

#### *Special Diagnostic Test*

Analysis of tissue for abnormal amounts of tin. See Gonzales et al., 1958.

#### *Recommended Threshold Limit*

(Tentative)

*Tin (inorganic compounds)*, 2 milligrams per cubic meter of air.

*Tin (organic compounds, as Sn)*, 0.1 milligram per cubic meter of air; should be reduced when also absorbed percutaneously.

### Potential Occupational Exposures

Babbitt metal (tin, copper, antimony) makers	Pigment makers
Bactericide workers	Plasticizer makers
Brass (essentially copper and zinc) founders	Plastic workers
Britannia metal (tin, copper, antimony) makers	Putty makers
Bronze (tin, copper) founders	Rodenticide workers
Ceramic makers	Solder makers
Dye makers	Sugar processors
Dyers	Textile makers
Fungicide workers	Textile printers
Paper makers, sensitized	Tin ore smelters
Perfume makers	Tin platers
Pewter (tin with lead, brass or copper) makers	Tin workers
	Type metal (lead, antimony, tin) makers

### References

BARNES, J. M. AND STONER, H. B.: Toxic properties of some dialkyl and trialkyl tin salts. *Brit. J. Indust. Med.* 15: 15, 1958.

GONZALES, T. A.; VANCE, M.; HELPERN, M., AND UMBERGER, C. J.: *Legal Medicine; Pathology and Toxicology*. 2nd ed. Appleton-Century-Crofts, New York, 1954.

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LYLE, W. H.: Lesions of the skin in process workers caused by contact with butyl tin compounds. *Brit. J. Indust. Med.* 15: 193, 1958.

PENDERGRASS, E. P. AND PRYDE, A. W.: Benign pneumoconiosis due to tin oxide. A case report with experimental investigation of the radiographic density of the tin oxide dust. *J. Indust. Hyg. & Toxicol.* 30: 119, 1948.

ROBERTSON, A. J.: Pneumoconiosis due to tin oxide. In King, E. J. and Fletcher, C. M. (editors): *Industrial Pulmonary Diseases*. Symposium, Postgraduate Medical School of London, 18-20 September 1957 and 25-27 March 1958. J. & A. Churchill, London, 1960.

ROBERTSON, A. J.; RIVERS, D.; NAGELSCHMIDT, G., AND DUNCUMB, P.: Stannosis; benign pneumoconiosis due to tin dioxide. *Lancet* 1: 1089, 1961.

### (177) Titanium and Compounds

#### Harmful Effects

*Local* High concentrations of titanium dioxide dust may produce irritation of respiratory tract. Titanium tetrachloride has a highly corrosive action upon skin and mucous membranes.

*Route of Entry.* Inhalation of dust or fume.

*Systemic* Titanium dioxide fume may produce metal fume fever. Titanium tetrachloride fume, when inhaled, produces severe lung irritation often resulting in pulmonary edema.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

(Titanium dioxide) 15 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Abrasive makers	Pearl makers
Cemented carbide makers	Pigment makers
Ceramic makers	Porcelain enamel makers
Cermet makers	Rayon makers
Corrosion inhibitor makers	Refractory material makers
Cosmetic makers	Resin makers
Electrode makers	Rubber makers
Electronic equipment makers	Shoe whitener makers
Flameproofers	Smoke screen makers
Foundry workers	Steel workers
Gem makers	Surfactant makers
Glass makers	Surgical instrument makers
Incandescent lamp makers	Tannery workers
Ink makers	Titanium alloy makers
Lacquer makers	Titanium metal refiners
Linoleum makers	Titanium workers
Nuclear steel makers	Vacuum tube makers
Painters	Varnishers
Paint makers	Waterproofing makers
Paper makers	Welding rod makers

### *Reference*

LAWSON, J. J.: The toxicity of titanium tetrachloride. *J. Occup. Med.* 3: 7, 1961.

### (178) Toluene

*toluol, methylbenzene, phenylmethane, methylbenzol*

### *Harmful Effects*

*Local* Liquid or vapor is primary irritant of skin, eyes, and mucous membranes of upper respiratory tract. Small corneal vacuoles have been produced by mixture of substances containing toluene.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid leading to systemic toxicity is improbable.

*Systemic* Primary effect of both acute and chronic exposures is central nervous system depression. Symptoms and signs include headache, dizziness, weakness, fatigue, paresthesia, disturbance of coordination and equilibrium, insomnia, and loss of consciousness. Onset and severity of symptoms will depend upon degree and type of exposure. Hematologic effects are not prominent; however, temporary and slight lymphocytosis has occasionally been observed.

### *Special Diagnostic Tests*

Analysis of urine for hippuric acid, and of blood for toluene. See Von Oettingen, 1958, and Gerarde, 1960.

### *Recommended Threshold Limit*

200 parts per million parts of air by volume or 750 milligrams per cubic meter of air

### *Potential Occupational Exposures*

Benzaldehyde makers	Pesticide workers
Benzoic acid makers	Petroleum refinery workers
Detergent makers	Printers
Drug makers	Resin workers
Dye makers	Rubber cement makers
Enamel makers	Saccharin makers
Explosive makers	Solvent workers
Gasoline blenders	Stainers
Gum processors	Stain makers
Histology technicians	Tannery workers
Ink makers	Textile workers
Laboratory workers, chemical	Thermometer makers, vapor pressure
Lacquerers	Toluene workers
Lacquer makers	Toluidine makers
Leather workers	Trinitrotoluene makers
Oil processors	Varnish makers
Painters	Vinyltoluene makers
Paint makers	Wax makers
Paint thinner makers	
Perfume makers	

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## (179) Toluene Diisocyanate

*2,4-toluene diisocyanate, TDI**Harmful Effects*

*Local* TDI vapor is highly irritating to eyes, nose, and throat, and produces conjunctivitis and coryza-like symptoms. Although TDI liquid is mildly irritating to skin, dermatitis is rare. Continued contact may darken and harden skin.

*Route of Entry* Inhalation of vapor.

*Systemic* Pulmonary irritation, and in some cases pulmonary sensitization, may cause nonproductive cough, wheezing, shortness of breath, and tightness of chest. Diagnoses of bronchitis and bronchial asthma are frequently made.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

(2,4-isomer) 0.02 part per million parts of air by volume or 0.14 milligram per cubic meter of air.

*Potential Occupational Exposures*

Abrasion resistant rubber makers	Polyurethane foam makers
Adhesive workers	Polyurethane sprayers
Aircraft builders	Ship burners
Insulation workers	Ship welders
Isocyanate resin workers	Spray painters
Lacquer workers	Textile processors
Mine tunnel coaters	Toluene diisocyanate workers
Organic chemical synthesizers	Upholstery makers
Plastic foam makers	Wire coating workers
Plasticizer workers	

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(180) Toxaphene. *See* Pesticides Section

(181) Trichloroethylene

*ethinyl trichloride, ethylene trichloride, trichloroethene*

### *Harmful Effects*

**Local** Liquid or high concentration of vapor may irritate eyes. Repeated contact with liquid or high vapor concentrations can produce a dry, scaly, and fissured dermatitis.

**Routes of Entry** Inhalation of vapor; of lesser importance, percutaneous absorption of liquid.

**Systemic** Trichloroethylene has a narcotic effect on central nervous system. In acute intoxications from low concentrations, manifestations include drowsiness, giddiness, dizziness, vertigo, fatigue, headache, exhilaration, nausea, vomiting, and incoordination. A characteristic symptom is intolerance toward alcohol. High vapor concentrations also have a narcotic effect and can produce unconsciousness, convulsions, coma, and death from respiratory paralysis. Death can occur from primary cardiac failure, ventricular fibrillation, and anoxia secondary to tachypnea and impaired alveolar ventilation. Reported cases of pulmonary edema may have been due to phosgene and hydrochloric acid, which are liberated when trichloroethylene is decomposed by heat.

A great variety of chronic effects have been attributed to trichloroethylene, such as liver damage, neuritis, and neurotic symptoms. Indication of liver damage is usually limited to abnormal liver function tests, but cases of acute yellow atrophy have been reported. The latter may have been due to contaminants or decomposition products. Injury to optic and trigeminal nerves has been reported. Neurotic symptoms are more difficult to evaluate and are doubted by some investigators.

### *Special Diagnostic Tests*

Determination of urinary metabolites, particularly trichloroacetic acid and trichloroethanol. *See* Seto and Schultze, 1956, and Souček and Vlachová, 1960.

### *Recommended Threshold Limit*

100 parts per million parts of air by volume or 520 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Anesthetic gas makers

Disinfectant makers

Caffeine processors

Drug makers

Cleaners

Dry cleaners

Coating makers

Dye makers

Degreasers

Dyers

Electronic equipment cleaners	Paper cup makers
Electroplaters	Perfume makers
Fat processors	Petroleum refinery workers
Fumigant workers	Photographic plate cleaners
Galvanizers	Polish makers
Gas purifiers	Printers
Gas workers, illuminating	Resin workers
Glass cleaners	Rubber cementers
Glue workers	Rubber workers
Heat transfer workers	Shoe workers
Lacquerers	Soap makers
Lacquer makers	Solvent workers
Leather workers	Stainers
Mechanics	Stain makers
Metal burnishers	Textile cleaners
Metal cleaners	Tobacco denicotinizers
Metal polishers	Trichloroethylene workers
Metal scourers	Vacuum tube makers
Oil processors	Varnishers
Optical lens cleaners	Varnish makers
Organic chemical synthesizers	Veterinarians
Painters	Wax makers
Paint makers	Wool scourers
Paint remover makers	

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### (182) Tricresyl Phosphate *TCP, tritolyl phosphate*

The ortho-derivative is the most toxic of the three isomers of tricresyl phosphate. The meta- and para-isomers are relatively inactive, but may contain the ortho-isomer as a contaminant unless special precautions were taken during manufacture.

## Harmful Effects

*Local* Contact dermatitis.

*Routes of Entry* Inhalation of vapor or mist; ingestion, percutaneous absorption of liquid.

*Systemic* Neurologic effects are caused by inhibition of cholinesterase as well as by demyelination and include polyneuritis and flaccid or spastic paralysis of extremities, usually the lower limbs. Recovery from paralysis may not be complete. There may be nystagmus, dysarthria, and accommodation difficulties.

## Special Diagnostic Test

Cholinesterase activity of plasma. See Elkins, 1959.

## Recommended Threshold Limit

(Triorthocresyl phosphate) 0.1 milligram per cubic meter of air.

## Potential Occupational Exposures

Gasoline additive makers  
Gasoline blenders  
Hydraulic fluid workers  
Lead scavenger makers  
Lubricant additive workers  
Nitrocellulose workers  
Plasticizer workers

Polystyrene makers  
Polyvinyl chloride makers  
Solvent workers  
Surgical instrument sterilizers  
Tricresyl phosphate workers  
Waterproofing makers

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## (183) Trinitrotoluene

*TNT, sym.-trinitrotoluol, methyltrinitrobenzene*

## Harmful Effects

*Local* Contact dermatitis from allergic hypersensitization. May stain skin a light yellow color and discolor hair to a reddish blond.

*Routes of Entry* Inhalation of dust, fume, or vapor. Ingestion of dust or percutaneous absorption from dust.

*Systemic* Gastrointestinal symptoms often occur first and include nausea, vomiting, and anorexia. Severe liver injury may follow and progress to acute yellow atrophy and death. Oxygen-carrying capacity of the blood is reduced through two mechanisms, namely, red blood corpuscle hemolysis, and formation of methemoglobin. Cyanosis, especially of lips, is a common finding. Breathlessness, weakness, and malaise may be present. Aplastic anemia has been reported to follow exposure to trinitrotoluene.

### *Special Diagnostic Test*

Qualitative and quantitative analyses of urine for trinitrotoluene and its metabolites. See Von Oettingen, 1958. The Webster test can be used to detect trinitrotoluene on skin or in clothing. See Norwood, 1943.

### *Recommended Threshold Limit*

1.5 milligrams per cubic meter of air. Should be reduced when also absorbed percutaneously.

### *Potential Occupational Exposures*

Demolition workers	Photographic chemical makers
Dye intermediate makers	Trinitrotoluene workers
Explosive fillers	

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## (184) Turpentine

*gum turpentine; oil of turpentine; spirit of turpentine; turps; gum spirit, derived from pine resin; wood turpentine, derived from pine stumps or sulfate wood pulp waste*

### *Harmful Effects*

*Local* Liquid may produce contact dermatitis from primary irritation as well as allergic hypersensitivity. High concentrations of vapor are irritating to eyes, nose, and throat.

*Routes of Entry* Inhalation of vapor; percutaneous absorption of liquid.

*Systemic* Headache, anorexia, gastritis, anxiety, excitement, mental confusion, tinnitus, bronchitis, and toxic nephritis.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

100 parts per million parts of air by volume or 560 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Art glass workers	Pine oil makers
Belt dressing makers	Resin makers
Camphor makers	Rubber reclaim workers
Drug makers	Rubber workers
Furniture polishers	Shoe polish makers
Furniture polish makers	Solvent workers
Ink makers	Stainers
Insecticide makers	Stain makers
Lacquerers	Stove polishers
Lacquer makers	Stove polish makers
Leather polish makers	Turpentine workers
Lithographers	Varnish workers
Oil additive makers	Wax makers
Paint workers	

## (185) Uranium and Compounds

Although uranium and its salts are highly toxic materials, poisoning attributable to their use in industry has not been a serious problem in this country.

### *Harmful Effects*

*Local* Principal skin hazard in handling uranium metal is exposure of hands to beta radiation.

*Route of Entry* Inhalation of fume, dust or gas. The following uranium salts are reported to be capable of penetrating intact skin.

Uranyl nitrate	Sodium diuranate
Uranyl fluoride	Ammonium diuranate
Uranium pentachloride	Uranium hexafluoride
Uranium trioxide	

*Systemic* Uranium and its salts, when absorbed into body, are highly toxic and may cause hepatic degeneration and chronic nephritis. Uranium

hexafluoride fumes, when inhaled, may produce a severe chemical pneumonitis. Prolonged inhalation of significant quantities of uranium, its salts, or its decay product, radon gas, may play an important role in causation of lung cancer.

### *Special Diagnostic Test*

Analysis of urine for uranium. See Elkins, 1959.

### *Recommended Threshold Limit*

*Uranium (soluble compounds)*, 0.05 milligram per cubic meter of air.

*Uranium (insoluble compounds)*, 0.25 milligram per cubic meter of air.

### *Potential Occupational Exposures*

Atomic bomb workers	Uranium hexafluoride makers
Ceramic makers	Uranium millers
Glass makers	Uranium miners
Hydrogen bomb workers	Uranium paint makers
Nuclear reactor workers	Uranium processors
Photographic chemical makers	Uranium workers
Pigment makers	Vanadium millers
Uranium alloy makers	Vanadium miners

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## (186) Vanadium

### *Harmful Effects*

*Local* Irritant to mucous membranes of eyes, nose, throat, and upper respiratory tract. Vanadium tetrachloride has been reported to be a skin irritant. Greenish discoloration of tongue is common among vanadium workers but is of no known toxicologic significance.

*Route of Entry* Inhalation of dust.

*Systemic* Reduction of serum cholesterol levels. Pulmonary irritation, possibly pneumonitis.

### *Special Diagnostic Test*

Analysis of urine for vanadium. See Lewis, 1959, part 1.

### *Recommended Threshold Limit*

*Vanadium pentoxide dust*, 0.5 milligram per cubic meter of air.

*Vanadium pentoxide fume*, 0.1 milligram per cubic meter of air.

### Potential Occupational Exposures

Alloy makers	Petroleum refinery workers
Boiler cleaners	Photographic chemical makers
Ceramic makers	Textile dye workers
Dye makers	Uranium millers
Dyers	Vanadium alloy makers
Ferrovanadium workers	Vanadium millers
Glass makers	Vanadium miners
Ink makers	Vanadium workers
Organic chemical synthesizers	

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### (187) Vinyl Chloride

*chloroethylene, chlorethane, monochloroethylene*

### Harmful Effects

*Local* Liquid is irritating to skin and eyes.

*Route of Entry* Inhalation of gas.

*Systemic* Gas is central nervous system depressant and produces dizziness and disorientation. Two deaths from occupational exposure to vinyl chloride have been recorded. No characteristic findings were noted at autopsy.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

500 parts per million parts of air by volume or 1,300 milligrams per cubic meter of air.

### Potential Occupational Exposures

Organic chemical synthesizers	Rubber makers
Polyvinyl resin makers	Vinyl chloride workers

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(188) Warfarin. *See* Pesticides Section

(189) Xylene

*xylol, dimethylbenzene*

### *Harmful Effects*

*Local* Xylene and its concentrated vapor are irritating to eyes, nose, and throat. Repeated contact of liquid with skin will produce a dry, scaly, and fissured dermatitis.

*Routes of Entry* Inhalation of vapor and, to a small but unimportant extent, percutaneous absorption of liquid.

*Systemic* Acute toxicity of inhaled xylene vapor is due to its vasodilatory and narcotic effects. Symptoms include flushing of face, headache, fatigue, confusion, paresthesias, dizziness, sleepiness, and unconsciousness. Chronic xylene poisoning probably does not occur.

### *Special Diagnostic Test*

None.

### *Recommended Threshold Limit*

200 parts per million parts of air by volume or 870 milligrams per cubic meter of air.

### *Potential Occupational Exposures*

Adhesive workers	Pathologists
Aviation gasoline workers	Pesticide workers
Bacteriologists	Petroleum refinery workers
Benzoic acid makers	Phthalic anhydride makers
Brake lining makers	Polyethylene terephthalate film makers
Catgut sterilizers	Protective coating workers
Color printers	Quartz crystal oscillator makers
Drug makers	Resin makers
Dye makers	Rubber cement makers
Enamel workers	Rubber workers
Histology technicians	Silk finishers
Ink makers	Solvent workers
Lacquerers	Stainers
Lacquer makers	Stain makers
Leather makers	Terephthalic acid makers
Lithographers	Vitamin makers
Microscopists	Xylene workers
Organic chemical synthesizers	
Painters	

## (190) Zinc and Compounds

*Harmful Effects*

*Local* Zinc chloride is extremely irritating to skin and may produce extensive ulceration; very irritating to eyes, nose, and throat. Perforation of nasal septum may be produced. Zinc chromate, zinc cyanide and zinc sulfate may cause dermatitis.

*Route of Entry* Inhalation of fume, dust, or vapor.

*Systemic* Inhalation of zinc chloride fumes may produce severe pneumonitis. Certain smoke-screening compounds produce upon ignition essentially zinc chloride and aluminum oxide. When inhaled, the zinc chloride in extremely high concentrations of finely divided particles will produce a chemical irritation of the upper respiratory tract; in the concentrations usually met with among military personnel, an insidious chemical pneumonitis has been reported to occur.

When metallic zinc is heated to a temperature near its boiling point, very finely divided zinc oxide fume is produced. Inhalation of freshly formed fumes may produce a brief, self-limiting illness known variously as *zinc chills*, *metal fume fever*, *brass chills*, and *brass founder's fever*. This condition is characterized by chills, fever, nausea, vomiting, muscular pain, dryness of mouth and throat, headache, fatigue, and weakness. There may also be a slight leukocytosis. These signs and symptoms usually abate in 12 to 24 hours with complete recovery following. Immunity from this condition is rapidly acquired if exposure occurs daily but is quickly lost during holidays or over weekends. Certain other metallic oxide fumes may cause this condition. These include the oxides of nickel, copper, magnesium, cadmium, iron, mercury, tungsten and titanium.

*Special Diagnostic Test*

None.

*Recommended Threshold Limit*

(Zinc oxide fume) 5 milligrams per cubic meter of air.

*Potential Occupational Exposures**Zinc*

Alloy makers	Metal cutters
Arc welders, electric	Metalizers
Brass foundry workers	Metal sprayers
Braziers	Printing plate makers
Bronze foundry workers	Roofing makers
Electric fuse makers	Zinc smelters
Electroplaters	Zinc workers
Galvanizers	

*Zinc compounds*

Activated carbon makers	Match makers
Adhesive makers	Mercerizers
Antistatic agent makers	Metal etchers
Battery makers, dry	Metal platers
Candle makers	Microscopists
Ceramic makers	Military personnel
Cosmetic makers	Organic chemical synthesizers
Crepe makers	Painters
Dental cement makers	Paint makers
Dentifrice makers	Paper makers
Deodorant workers	Petroleum refinery workers
Disinfectant makers	Pigment makers, steel
Dye makers	Polish makers, steel
Electroplaters	Railroad tie preservers
Embalmers	Rayon makers
Embalming fluid workers	Rubber makers
Enamel makers	Seed treaters
Feed additive makers	Silk makers
Fungicide workers	Smoke screen makers
Galvanizers	Soap makers
Gelatin makers	Solder flux makers
Glass etching agent makers	Taxidermists
Glue makers	Textile crimpers
Illuminating gas workers	Textile fireproofers
Ink makers	Textile makers
Insecticide workers	Textile mordanters
Iron copper-platers	Textile sizers
Linoleum makers	Textile weighters
Lithopone makers	Varnish makers
Lumber fireproofers	Wood preservative workers
Magnesium oxide cement makers	

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## (191) Zirconium Compounds

*Harmful Effects*

**Local** Certain of the zirconium salts used in antiperspirant preparations have been reported to produce granulomatous lesions in axilla.

*Route of Entry* Inhalation of dust or fume.

*Systemic* No systemic effects in workers have been reported.

### Special Diagnostic Test

None.

### Recommended Threshold Limit

Zirconium compounds (as Zr), 5 milligrams per cubic meter of air.

### Potential Occupational Exposures

Abrasive makers	Metallurgists
Alloy makers	Nuclear reactor workers
Arc lamp makers	Paint makers
Ceramic workers	Phosphor makers
Cermet makers	Photographic illuminant makers
Crucible makers	Pigment makers
Deodorant makers	Polish makers
Drug makers	Pottery makers
Dye makers	Rayon spinneret makers
Enamel makers	Refractory material makers
Explosive makers	Steel makers
Foundry workers	Tannery workers
Furnace lining makers	Textile waterproofers
Gem makers	Vacuum tube makers
Glass makers	Varnish makers
Incandescent lamp makers	Waterproofers
Lacquer makers	Zirconium workers
Lubricant makers	

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## • section VII

### PESTICIDES

ROY L. GIBSON, M.D., AND THOMAS H. MILBY, M.D.

The beneficial effects of pesticides in disease control and crop production are well recognized. In many areas of the world the chemical eradication of insect and rodent vectors has partially or completely eliminated such diseases as malaria, yellow fever, dengue, and plague. Populations tend to increase with the disappearance of such potentially lethal diseases, and the current population explosion is in part the result of these pest control measures.

Growing populations require more land for habitation and food for consumption; therefore, optimum crop production becomes a necessity. The shrinking-world phenomenon forces the public health worker and others to become acquainted not only with an increasing variety of pests of all kinds but also with the new pesticides which are being developed to cope with the problem. Pesticides popular today may be of little use tomorrow because of the development of pest resistance to these chemicals. Thus, man is confronted with thousands of these products, many of which may impose a human liability if exposure is sufficient and uncontrolled.

For the purpose of this presentation, the pesticides are divided into five major groups that denote their principal proposed use. These groups include insecticides, rodenticides, fungicides, herbicides, and fumigants. The insecticides are further categorized according to chemical configuration into chlorinated hydrocarbons, phosphate esters, and miscellaneous insecticides.

For clarity and to facilitate presentation, the pesticides are generally designated by the term or terms most commonly appearing in the medical literature. Pesticide solvents such as kerosine, naphtha, toluene and xylene, which are not presented here, are included in the section on Chemical Hazards.

No attempt is made to present the clinical effects of all pesticides currently used. Such information may be found in more extensive treatises on the subject. Specific toxicologic information is lacking for many of these compounds, and others are not of sufficient toxicity or use to pose significant health problems.

Hazardous exposures may occur in both occupational and nonoccupational pursuits. Manufacturers, spray pilots, crop workers, farmers, nursery workers, seed-treaters, soil fumigators, exterminators, and others engaged in the development, manufacture, distribution, and use of these chemicals are subject to intoxication if proper precautions are not observed. Such nonoccupational activities as gardening, yard maintenance, and camping expose millions more to these agents. An understanding of their harmful effects is therefore essential.

### Insecticides

#### *Chlorinated Hydrocarbons*

All chlorinated hydrocarbon insecticides, in general, produce similar physiologic effects in man, and consequently their clinical characteristics can be discussed collectively. However, they vary in their ability to cause clinical symptoms by virtue of differences in absorption and excretion. Intoxication may result from ingestion, inhalation, or percutaneous absorption. Headache, loss of appetite, nausea, vomiting, dizziness, tremors, convulsions, and coma occur following excessive exposure.

The insecticide or its derivatives can often be demonstrated in urine, stomach contents or body tissues, especially fat. Some of the chemicals in this group—for example, chlordane and benzene hexachloride—have been reported to cause dermatitis either by primary irritation or hypersensitization.

The chlorinated hydrocarbons of greatest significance and their threshold limit values, in milligrams per cubic meter of air, follow.

Aldrin	0.25 mg. per cu. m.*
Benzene hexachloride	Not established.
Chlordane	0.5 mg. per cu. m.
DDT (chlorophenothane)	1 mg. per cu. m.*
Dieldrin	0.25 mg. per cu. m.*
Endrin	0.1 mg. per cu. m. (tentative)*
Heptachlor	0.5 mg. per cu. m. (tentative)
Lindane (gamma-benzene hexachloride)	0.5 mg. per cu. m.
Methoxychlor	15 mg. per cu. m.
Terpene polychlorinates	Not established.
Toxaphene (chlorinated camphene, 60 percent).	0.5 mg. per cu. m.

\*Should be reduced when also absorbed percutaneously.

#### *Phosphate Esters*

The phosphate ester insecticides are characterized by the similarity of their structural relationship—they are phosphoric acid derivatives—and by their identical mechanism of toxic action. They differ widely, however, in inherent toxicity and, to some extent, in rate of absorption and excretion.

These compounds are readily absorbed through the intact skin. They may also be absorbed following ingestion or inhalation of contaminated materials.

The organic phosphorus compounds act as irreversible inhibitors of cholinesterase, thereby allowing the accumulation of large amounts of acetylcholine. When a critical level of cholinesterase depletion is reached, usually about 20 percent of normal, symptoms and signs of poisoning become manifest. Symptoms may include blurred vision, weakness, nausea, headache, abdominal cramps, chest discomfort, and diarrhea. Signs may include miosis, muscle twitching, salivation, sweating, tearing, cyanosis, convulsions, and coma. Peripheral nerve damage has been reported as an occasional sequela of poisoning by certain organic phosphates.

Diagnosis is based upon the following criteria: a definite history of exposure of six hours or less before the onset of symptoms, clinical evidence of diffuse parasympathetic stimulation, and depression of plasma and red blood cell cholinesterase. The urinary excretion of p-nitrophenol may be useful in the diagnosis of parathion or EPN intoxication.

The more commonly encountered phosphate ester insecticides together with their threshold limit values, in milligrams per cubic meter of air, are grouped according to toxicity in the following list.

*Highly toxic:*

Dematon (Systox <sup>R</sup> ) -----	0.1 mg. per cu. m.* (tentative)
Ethyl p-nitrophenyl thionobenzene phosphonate (EPN). -----	0.5 mg. per cu. m.*
Guthion <sup>R</sup> -----	Not established.
Hexaethyl tetraphosphate (HETP) -----	Not established.
Methyl parathion -----	Not established.
Octamethyl pyrophosphoramide (OMPA). -----	Not established.
Parathion -----	0.1 mg. per cu. m.*
Phosdrin <sup>R</sup> -----	0.1 mg. per cu. m.*
Pyrazoxon <sup>R</sup> -----	Not established.
Tetraethyl dithionopyrophosphate (TEDP). -----	0.2 mg. per cu. m.*
Tetraethyl pyrophosphate (TEPP) -----	0.05 mg. per cu. m.*
Thimet <sup>R</sup> -----	Not established.
Trithion <sup>R</sup> -----	Not established.

*Moderately toxic:*

Diazinon <sup>R</sup> -----	Not established.
Dimethyldichlorvinyl phosphate (DDVP). -----	1 mg. per cu. m.* (tentative)
Ethion -----	Not established.

\*See footnote at end of table.

*Slightly toxic:*

Chlorthion <sup>R</sup>	-----	Not established.
Dipterex <sup>R</sup>	-----	Not established.
Malathion	-----	15 mg. per cu. m. *

\*Should be reduced when also absorbed percutaneously.

*Miscellaneous Insecticides*

Although the newer synthetic pesticides previously discussed in this section are becoming increasingly popular, the following compounds continue to find significant usage.

*Lead arsenate and arsenite*—These compounds enter the body by inhalation, ingestion, or percutaneous absorption. Signs and symptoms of poisoning are similar to those characteristic of lead or arsenic intoxication. Acute symptoms includes nausea, vomiting, abdominal pain, diarrhea, muscle cramps, excitation, and disorientation. Chronic poisoning is manifested by anorexia, weakness, weight loss, pallor, colic, diarrhea, peripheral neuritis, hepatitis, and nephritis. A vesicular dermatitis has frequently been reported. The carcinogenic hazard from chronic arsenic exposure cannot be ignored. The recommended threshold limit value for lead arsenate is 0.15 milligram per cubic meter of air.

*Nicotine*—This extremely toxic alkaloid is capable of producing nervous system stimulation followed by severe nervous system depression. The effects may result from ingestion, inhalation, or rapid percutaneous absorption of the material. Analysis for urinary nicotine may aid in the diagnosis. The recommended threshold limit value is 0.5 milligram per cubic meter of air which should be reduced when also absorbed percutaneously.

*Pyrethrum*—This material is not particularly toxic; however, primary contact dermatitis and allergic skin and pulmonary reactions have occurred following minimal exposure to the dust. The recommended threshold limit value is 5 milligrams per cubic meter of air.

*Rotenone*—This plant extract is more toxic than pyrethrum but, as normally used, is not excessively hazardous. Contact dermatitis and numbness of the oral mucous membranes may follow sufficient exposure. The recommended threshold limit value is 5 milligrams per cubic meter of air.

### Rodenticides

Rodenticides of first importance include sodium fluoroacetate, strychnine, thallium sulfate, and warfarin. For information on rodenticides containing arsenic, barium, cyanide and phosphorus, reference may be made to the appropriate chemical in the section on Chemical Hazards.

*Sodium fluoroacetate (Compound 1080)*—This material may be absorbed through the skin and the respiratory and gastrointestinal tracts. Clinical manifestations include nausea, apprehension, cardiac irregularities, and con-

vulsions followed by central nervous system depression. The recommended threshold limit value is 0.05 milligram per cubic meter of air which should be reduced when also absorbed percutaneously.

*Strychnine*—Severe convulsions without loss of consciousness are characteristic of strychnine poisoning. Death is usually a result of asphyxia or involvement of vital brain centers. The compound may be identified in the urine soon after ingestion. The recommended threshold limit value is 0.15 milligram per cubic meter of air.

*Thallium sulfate*—Intoxication may follow ingestion or skin absorption. Acute poisoning is characterized by severe gastroenteritis following a latent period of 12 to 24 hours. Other effects may include liver and kidney damage, encephalopathy, neuritis, ataxia and alopecia. Recovery is slow. Thallium may be demonstrated in the urine. The recommended threshold limit value for soluble compounds of thallium is 0.1 milligram per cubic meter of air which should be reduced when also absorbed percutaneously.

*Warfarin*—Intoxication occurs following chronic ingestion of warfarin, and the following signs and symptoms due to inhibition of prothrombin formation and capillary fragility are observed: pallor, bleeding gums, nose bleeds, petechial rash, bruises, blood in the urine and stools, and shock. Laboratory studies reveal a prolonged prothrombin time. The recommended threshold limit value for warfarin is 0.1 milligram per cubic meter of air.

### Fungicides

The fungicides are a heterogeneous group of chemicals and, with the major exception of the dithiocarbamates, have been in use for many years. Many of the fungicides—formaldehyde, furfural, phenol, tetramethylthiuram disulfide and compounds of boron, chromium, copper, mercury, tin and zinc—some of which are also used as herbicides and insecticides, are discussed in the section on Chemical Hazards.

The dithiocarbamates include ferbam (ferric dimethyldithiocarbamate), ziram (zinc dimethyldithiocarbamate), maneb (manganous ethylene bis-dithiocarbamate), nabam (disodium ethylene bisdithiocarbamate) and zineb (zinc ethylene bisdithiocarbamate). Their chief adverse effects are irritation of the skin, eyes, and upper respiratory tract.

Threshold limit values, in milligrams per cubic meter of air, have been recommended for the following fungicides as shown.

Ferbam	-----	15 mg. per cu. m.
Formaldehyde	-----	6 mg. per cu. m.
Organic mercurials	-----	0.01 mg. per cu. m.*
Pentachlorophenol (PCP)	-----	0.5 mg. per cu. m.*
Sodium dichromate	-----	0.1 mg. per cu. m. (as CrO <sub>3</sub> )

\*Should be reduced when also absorbed percutaneously.

## Herbicides

Herbicides, or weed killers, may be classified as pesticide chemicals. They can kill plants on contact, or they can be translocated; that is, absorbed by one part of the plant and carried to other parts where they exert their primary toxic effect. Most of the commonly used herbicides—ammonium sulfamate, dalapon, phenoxyacetic acid derivatives, carbamate derivatives, petroleum oils, sodium borate, Crag<sup>R</sup> herbicide—have a low toxicity and have caused little difficulty among users.

Some herbicides pose more serious problems; for example, the central nervous system effects of maleic hydrazide or the methemoglobinemia and central nervous system depression of sodium chlorate. Pentachlorophenol, a metabolic stimulant, has been responsible for several deaths because of hyperthermia. Amino triazol has produced cancer in experimental animals, but there have been no untoward effects reported in man.

Herbicides with cutaneous effects include trichloroacetic acid, a corrosive irritant of the skin and mucous membranes; maleic hydrazide, a producer of allergic contact dermatitis; pentachlorophenol, a producer of a primary irritant type of contact dermatitis; and creosote, a primary irritant and photosensitizer.

Reference may be made to chemicals in the section on Chemical Hazards for the toxicity of the following herbicides: Arsenic trioxide and sodium arsenate (see Arsenic), copper sulfate (see Copper and Compounds), creosote compounds (see Cresol and Phenol), dinitrophenols (see Dinitrophenol), kerosine, and phenylmercuric acetate (see Mercury and Compounds).

Threshold limit values, in milligrams per cubic meter of air, have been recommended for the following herbicides as shown.

Ammonium sulfamate (Ammate <sup>R</sup> )	15 mg. per cu. m.
Crag <sup>R</sup> herbicide	15 mg. per cu. m.
2,4-D (2,4-dichlorophenoxyacetic acid)	10 mg. per cu. m.
Pentachlorophenol (PCP)	0.5 mg. per cu. m.*
Phenylmercuric acetate (PMA)	0.01 mg. per cu. m.* (for organic mercury)
2,4,5-T (2,4,5-trichlorophenoxyacetic acid).	10 mg. per cu. m. (tentative)

\*Should be reduced when also absorbed percutaneously.

## Fumigants

Fumigants are pesticides which may be applied in the solid, liquid, or gaseous state. A combination of high volatility with high pest toxicity is generally desired; however, compounds with low volatility may be preferred for soil fumigation. The possibility of excessive exposures exists wherever

fumigants are used, as in fumigating grains, soils, clothes, furs, homes, warehouses, barns, ships, mills, freight cars, and greenhouses.

Each of the following compounds has found use as a fumigant. Because they have other industrial applications as well, they are discussed individually in the section on Chemical Hazards.

Acrylonitrile	Methyl Bromide (see Bromine and Compounds)
Carbon Disulfide	Methylene Chloride
Carbon Tetrachloride	Methyl Formate
p-Dichlorobenzene (see Chlorinated Benzenes)	Naphthalene
Dioxane	Perchloroethylene
Ethylene Dibromide	Propylene Dichloride
Ethylene Dichloride	Sulfur Dioxide
Ethylene Oxide	Tetrachloroethane
Hydrogen Cyanide	Trichloroethylene

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## • section VIII

### PLASTICS AND SYNTHETIC RESINS

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Plastics were so named because at some stage in their manufacture they were soft enough to be formed into various shapes. Synthetic resins were named for their similarity to natural resins, such as rosin. Generally the words are interchangeable, although, for a specific plastic or resin, it is customary to use one in preference to the other; thus, epoxy resin rather than epoxy plastic.

There are a number of basic chemical or family groups of plastics and resins, several of which have been arbitrarily chosen for presentation. Each group may be classified as either thermosetting or thermoplastic. Thermosetting resins cure or harden under heat and cannot be reshaped subsequently. Examples are alkyds, allyls, aminos, diisocyanates, epoxies, phenolics, and polyesters. Thermoplastic resins can be softened by heat and reshaped repeatedly. Examples are acrylics, cellulosics, fluorocarbons, nylons, polyethylenes, polystyrenes, and vinyls.

Plastics and resins are made by polymerization or condensation. In polymerization, a large number of identical molecules, called monomers, unite to form a larger molecule, called a polymer. In condensation, a number of molecules, not necessarily of the same composition, unite to form a molecule dissimilar in composition to the components, with the liberation of water or other simple substances.

In addition to monomers and condensate components, many other materials are used in compounding plastics and resins; namely, accelerators, catalysts, copolymers, dyes, fillers (asbestos, diatomite, glass fiber, mica, quartz, sand and many other substances), mold lubricants, pigments, plasticizers, solvents, stabilizers, and ultraviolet absorbers.

The major hazard from the manufacture, curing, and processing of plastics and resins is contact dermatitis which may be due either to primary irritation or to allergic sensitization. As a rule, completely condensed or polymerized resins do not cause dermatitis. Frequent offenders are the catalysts,

low molecular weight polymers, monomers, and uncured condensate components. Accelerators, plasticizers, solvents, and other components may also cause dermatitis.

In addition to dermatitis, the manufacture, curing, and processing of some plastics and resins may be accompanied by systemic reactions such as bronchial asthma from tolylene diisocyanate and polymer fume fever from polytetrafluoroethylene. Most of the components that produce systemic effects are included in the section on Chemical Hazards under the following headings:

Acetic Acid	Ketones
Acetone (See Ketones)	Methyl Chloride
Allyl Alcohol	Organic-tin Compounds (See Tin)
Ammonia	Phenol
Amyl Acetate	Phosgene
Cresol	Phthalic Anhydride
Epichlorohydrin	Styrene
Ethyl Acetate	Tolylene Diisocyanate
Formaldehyde	Tricresyl Phosphate
Furfural	Vinyl Chloride
Hexamethylenetetramine	

In the following 14 major family groups, material is presented with regard to harmful effects, and potential occupational exposures. Because of the rapid changes in the plastics industry, it is understandable that the potential occupational exposures as given may require periodic revision. Basic information on chemical technology and production technics may be secured by referring to the *Modern Plastics Encyclopedia* issued annually as a part of the periodical, *Modern Plastics*.

### Thermosets

#### (1) *Alkyd Resins*

Some of the condensate materials can produce contact dermatitis; for example, phthalic and maleic anhydride.

#### *Potential Occupational Exposures*

Alkyd resin makers  
Lacquer makers

Textile finishers

#### (2) *Allyl Resins*

Contact dermatitis has occurred from the monomers as well as their precursors, allyl alcohol and diglycochlorformate. The use of phosgene to make diglycochlorformate can produce pulmonary edema. The organic peroxide catalysts used to cure the prepolymers can produce contact dermatitis.

*Potential Occupational Exposures*

Aircraft part makers	Electric part makers
Allyl prepolymer makers	Missile component makers
Decorative laminators	

**(3) Amino Resins***urea-formaldehyde resins, melamine-formaldehyde resins*

The most common dermatitis-producing component is formaldehyde. It may be encountered in the production of the amino resins or may be released subsequently from improperly cured urea-formaldehyde or melamine-formaldehyde crease-resistant fabrics and produce allergic contact dermatitis and conjunctivitis. Formaldehyde reaction products, for example, dimethylol urea, in textile finishes can also act as sensitizers. Hexamethylenetetramine, a formaldehyde liberator, is used as a stabilizer for urea-formaldehyde molding compounds and can also produce dermatitis.

*Potential Occupational Exposures*

Adhesive makers	Foundry workers
Aerosol dispenser makers	Furniture makers
Automobile ignition makers	Gluers
Button makers	Lace tenters
Crease-resistant textile finishers	Paper treaters
Cutlery handle makers	Plywood makers
Decorative tabletop makers	Shirt makers
Dish makers	Soap makers
Dress makers	

**(4) Diisocyanate Resins***polyurethane resins*

The diisocyanates, usually tolylene diisocyanate and diphenyl-methane diisocyanate, are irritating to the skin, and some of the tertiary amine catalysts are strong alkaline irritants, but dermatitis in polyurethane foam production is rare. Tolylene diisocyanate is irritating to the eyes and respiratory tract, and can produce allergic bronchial asthma. Cutaneous spills of exothermally reacting polyurethane foam can produce burns.

*Potential Occupational Exposures*

Adhesive makers	Float makers
Aircraft part makers	Freezer makers
Boat makers	Life preserver makers
Crash pad fillers	Printing roll makers
Cushion makers	Refrigerator makers

### (5) *Epoxy Resins*

Both epichlorohydrin and bisphenol, used to make the epoxy resins, can produce dermatitis. The liquid epoxy resins are moderate irritants and sensitizers. The solid epoxy resins are less likely to affect the skin, but their solvents, such as ketones, esters and ethers, frequently produce excessive drying, scaling, and fissuring of the skin. The resins may also contain plasticizers such as dibutyl phthalate and tricresyl phosphate, and reactive diluents (various glycidyl ethers), all of which can produce contact dermatitis. The most frequent dermatitis-producing components are the aliphatic polyamine curing agents; for example, diethylenetriamine and triethylene-tetramine. These are highly alkaline, primary irritants and sensitizers. Aromatic amine curing agents such as metaphenylenediamine, and anhydride curing agents such as phthalic anhydride, can also produce contact dermatitis. Glass fiber used in making laminates frequently produces a mechanical irritation of the skin accompanied by considerable pruritus. Cured epoxy resins, especially if heat-cured, are usually inert, but subsequent tooling operations may be associated with dermatitis from glass fiber laminate particulates or possibly from resin-decomposition products.

Inhalation of the vapors of aliphatic polyamines can produce asthma-like symptoms and urticaria. The amines and their vapors are irritating to the conjunctiva.

#### *Potential Occupational Exposures*

Adhesive makers	Electron microscopists
Aircraft panel makers	Gluers
Appliance sprayers	Highway maintenance workers
Automobile body repairmen	Laminators
Automobile prototype makers	Paint makers
Body solder makers	Paint sprayers
Brick masons	Pattern makers
Cement patchers	Tank coaters
Electric equipment makers	Tile setters
Electricians	Tool and die makers

### (6) *Phenolic Resins*

*phenol-formaldehyde resins, cashew-nut-shell-liquid formaldehyde resins*

Contact dermatitis can result from any of the phenolic components such as phenol, cresol, resorcinol, and cashew nut shell liquid; aldehyde components such as formaldehyde, paraformaldehyde, and furfural; or catalysts such as ammonia and hexamethylenetetramine. The most frequent offenders are formaldehyde and hexamethylenetetramine, which are irritants as well as

sensitizers. Mineral oil, which surrounds the resin in the curing ovens, can produce folliculitis. Irritation of the eyes and upper respiratory tract are occasionally produced by formaldehyde vapors from the various operations associated with mixing, extrusion, molding, laminating, and casting.

#### *Potential Occupational Exposures*

Abrasive wheel makers	Nose cone makers
Brake lining makers	Particle board makers
Cabinet makers	Plywood makers
Decorative laminators	Resin extrusion workers
Electric circuit printers	Sandpaper makers
Electric component makers	Shell molders
Foundry workers	Sports equipment makers
Glass wool insulation makers	Television cabinet makers
Headphone makers	Toilet seat makers
Lacquer makers	Toy makers
Luggage makers	Varnish makers
Missile blast tube makers	

#### *(7) Polyester Resins*

Phthalic or maleic anhydride used in the manufacture of polyester monomer can produce dermatitis. The polyester monomer, modifiers such as styrene and methyl methacrylate, accelerators such as cobalt naphthenate and dimethyl aniline, catalysts such as organic peroxides, and plasticizers such as tricresyl phosphate and dibutyl phthalate can also produce contact dermatitis. The glass fiber used for lamination produces a pruritic, mechanical dermatitis.

Symptoms of headache, nausea, vomiting, and anorexia from styrene inhalation are occasionally encountered. Dimethyl aniline is a central nervous system depressant and may be absorbed through the skin as well as by inhalation.

#### *Potential Occupational Exposures*

Adhesive makers	Food wrapper makers
Artificial limb makers	Geodesic dome makers
Automobile body repairmen	Glass fiber sheeting makers
Automobile glass fiber body makers	Ignition part makers
Boat makers	Lampshade makers
Body solder makers	Laundry tub makers
Chair makers	Luggage makers
Electric equipment makers	Pipe makers
Fishing rod makers	Swimming pool makers
	Translucent panel makers

## Thermoplastics

### (8) *Acrylic Resins*

The most widely used monomer, methyl methacrylate, is a sensitizer. The catalyst benzoyl peroxide can also produce dermatitis. Inhalation of methyl methacrylate vapor can produce irritability, headache, anorexia, somnolence, and hypotension. Solvents used to make solution polymers can produce a dry, scaly, and fissured dermatitis.

#### *Potential Occupational Exposures*

Acrylic emulsion makers  
Acrylic molding bead makers  
Acrylic resin casters

Acrylic solution polymer makers  
Dental technicians

### (9) *Cellulosics*

*cellulose nitrate, cellulose acetate, cellulose acetate butyrate, and cellulose propionate*

Dermatitis occurs from mineral acids and alkalis used to treat the cellulose pulp. Acetic acid used in the production of cellulose acetate has caused mucosal irritation and dental erosion. Dermatitis may occur from plasticizers such as tricresyl phosphate, dibutyl phthalate, and dimethyl phthalate, and from solvents such as alcohol, acetone, ethyl acetate, and amyl acetate in the production of celluloid or pyroxylin from scraps of cellulose nitrate or cellulose.

#### *Potential Occupational Exposures*

Cellulose derivative makers  
Cellulose plastic compounders

Synthetic fiber makers

### (10) *Fluorocarbons*

*polytetrafluoroethylene*

Inhalation of freshly generated polytetrafluoroethylene (Teflon) dust, especially when heated, can produce a self-limited condition known as polymer fume fever, which is manifested by chills, fever, and aches in muscles and joints. Inhalation of thermal decomposition products of polytetrafluoroethylene can also irritate the respiratory tract and produce pulmonary edema.

#### *Potential Occupational Exposures*

Bearing makers  
Electric insulation makers  
Gasket makers

Machinists  
Pump diaphragm makers  
Tubing makers

**(11) Nylons***polyamides*

No harmful effects have been reported in manufacturing or processing.

**(12) Polyethylenes**

Dermatitis is rare but has occurred from contact with the resin before it has completely polymerized.

*Potential Occupational Exposure*

Ethylene polymer makers

**(13) Polystyrenes**

The styrene monomer is a skin irritant and produces systemic symptoms of headache, nausea, vomiting, and anorexia. Organic peroxide catalysts and polymerization solvents can produce contact dermatitis. Central nervous system damage resulting in dizziness, staggering gait, and death has occurred from inhalation of methyl chloride, which may be released in the fabrication of polystyrene foam.

*Potential Occupational Exposures*

Polystyrene foam fabricators

Styrene polymer makers

**(14) Vinyl Plastics***polyvinyl chloride, polyvinyl acetate, polyvinyl alcohol*

Dermatitis is rarely encountered although the monomers are irritants. Plasticizers and stabilizers occasionally cause contact dermatitis.

*Potential Occupational Exposures*

Leather makers, artificial

Vinyl polymer makers

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## • section IX

### PHYSICAL HAZARDS

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The hazards considered are those associated with exposure to the following agents:

(1) Ultraviolet, Infrared, and Microwave Radiation	(4) Abnormal Temperature
(2) Ionizing Radiation	(5) Defective Illumination
(3) Abnormal Air Pressure	(6) Noise and Vibration

Some of the hazards are encountered only in specific occupational situations, while others may be present in numerous working environments.

The physical agents discussed usually produce tissue injury by imparting energy in a harmful form to the tissue. This energy may be great and applied to the whole surface of the body such as in the instance of increased air pressure. On the other hand the energy may be small but applied at the cellular level such as noise or at the intracellular level as is the case with ionizing radiation.

Although a thorough investigation of the effect on man of devices employing the laser (light amplification by stimulated emission of radiation) has not been reported, the subject is of sufficient occupational health importance to merit brief mention. With the use of electric energy, the laser creates a high light intensity having acute biologic significance when the direct or optically reflected rays impinge on biologic tissue. Much work is being done on the development of devices, or in attempts to improve their efficiency and mode of operation, in such diverse fields as tracking and ranging (radar), communication, high temperature research, micromeasurement, microsurgery, biologic tissue removal or stimulation, high resolution spectroscopy, and microwelding. Sufficient experimental work on animals has been performed to indicate that persons working with laser light sources should be cognizant of the potentially hazardous nature of their occupation.

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### (1) Ultraviolet, Infrared, and Microwave Radiation

The continuum of energy extending from the long wave lengths of radio to the short wave lengths of cosmic radiation has been designated the electromagnetic spectrum. In between are television, microwaves, infrared rays, visible light rays, ultraviolet rays, X-rays, and gamma rays. The visible or light waves vary in length from 4,000 to 7,600 Angstrom units, with the ultraviolet rays below the 4,000 Å lower limit and the infrared and microwaves, respectively, above the 7,600 Å upper limit. (10 Å equal 1 millimicron or one millionth of a millimeter.) Ultraviolet and infrared rays, and microwaves, are invisible radiation. Because of their physical characteristics, alpha particles, beta particles, protons, neutrons, and positrons are classified under corpuscular (or particulate) radiations rather than under the radiations of the electromagnetic wave type. An electromagnetic wave, as the name implies, involves both an electric field and a magnetic field, each being measurable. All electromagnetic oscillations in free space travel with the speed of light, and cease locally when the source is turned off or removed. The power density is inversely proportional to the square of the distance from the source.

#### *Ultraviolet Radiation*

The sunburn spectrum of sunlight is in the ultraviolet zone, 2,900–3,100 Angstrom units, with a peak at about 3,000. Repeated exposures of fair-skinned individuals to the sunburn spectrum results in actinic skin. This is manifested by a dry, brown, inelastic and wrinkled skin. On the face, there are telangiectases, and on the nape, the neck movements produce lines in an angular pattern. Sailor's, farmer's, or fisherman's skin are names given to actinic skin, which bespeak their occupational origin. Oil field, pipeline, and construction workers also develop this condition. Actinic skin is not harmful in itself, but is a warning to susceptible individuals who tan poorly that certain conditions may develop such as senile keratoses, squamous cell epitheliomas and basal cell epitheliomas.

The ultraviolet light generated during welding can cause keratoconjunctivitis and sunburn of the exposed skin. The welder's helper, whose protection is more likely to be deficient, more often suffers the damage. Some of the newer welding processes, such as inert-gas, metal-arc welding with consumable electrodes, produce high intensities of ultraviolet light, and consequently have caused a marked increase in frequency and severity of eye and skin burns.

Photosensitizing agents have action spectra which are frequently in the ultraviolet range. Many plants such as figs, limes, parsnips, and pink-rot celery carry photosensitizing chemicals. These are believed to be furocoumarins and psoralens. The signs upon contact are those of an exaggerated sunburn, and blisters are frequently present. The most important industrial photosensitizer is coal tar, with an action spectrum in the visible light range. The increased incidence of skin cancer in coal tar workers is not only due to carcinogens, but also to repeated bouts of photosensitization.

Ultraviolet light can also act in a nonspecific manner similar to trauma in producing lesions of herpes simplex and chronic discoid lupus erythematosus.

Incandescent and fluorescent lamps used for general lighting purposes emit little or no ultraviolet radiation and are generally considered harmless. A rare reaction to fluorescent lighting has been observed in individuals who are sensitive to visible light. This sensitivity is manifested by urticaria (urticaria solaris) or by erythema and edema (erythema solare perstans) of exposed areas.

*Potential occupational exposures*—Occupations potentially associated with ultraviolet radiation exposures include the following.

Aircraft workers	Iron workers
Barbers	Lifeguards
Bath attendants	Lithographers
Brick masons	Metal casting inspectors
Burners, metal	Miners, open pit
Cattlemen	Nurses
Construction workers	Oil field workers
Cutters, metal	Pipeline workers
Drug makers	Plasma torch operators
Electricians	Railroad track workers
Farmers	Ranchers
Fishermen	Road workers
Food irradiators	Seamen
Foundry workers	Skimmers, glass
Furnace workers	Steel mill workers
Gardeners	Stockmen
Gas mantle makers	Stokers
Glass blowers	Tobacco irradiators
Glass furnace workers	Vitamin D preparation makers
Hairdressers	Welders
Herders	

### *Infrared Radiation*

The action of infrared rays is thermal, and fortunately such action gives a warning in the skin. However, in the eye there is no warning, and damage

may be produced by amounts of energy which will not burn the skin. The classic eye lesion after many years of exposure is posterior cataract, sometimes called glassblower's cataract, but this has not been reported in the United States. Mild exposures to infrared can cause eye fatigue and headaches. On the skin, infrared radiation produces a thermal burn.

Both infrared and ultraviolet radiation are present in some industrial exposures—for example, oxacetylene and electric welding; cupola, open-hearth, and electric furnace operations and foundry pouring; and glass blowing.

*Potential occupational exposures*—Occupations potentially associated with infrared radiation exposures include the following.

Bakers	Heat treaters
Blacksmiths	Iraser operators (infrared amplification by stimulated emission of radiation)
Braziers	
Chemists	
Cloth inspectors	Iron workers
Cooks	Kiln operators
Dryers, lacquer	Motion picture machine operators
Electricians	Plasma torch operators
Firemen, stationary	Skimmers, glass
Foundry workers	Solderers
Furnace workers	Steel mill workers
Gas mantle hardeners	Stokers
Glass blowers	Welders
Glass furnace workers	

### *Microwave Radiation (radar)*

Microwave radiation or microwave energy includes electromagnetic frequencies ranging from about 300 megacycles to more than 30,000 megacycles per second. This form of energy is normally propagated in the atmosphere from rotating antennas associated with search radar and from stationary types associated with tracking radar, radio relay links, and television transmitters. Industrial and medical apparatus utilizing the heating effect of this energy include radio frequency ovens and diathermy devices.

This energy, when propagated, is categorized into two discrete modes known as continuous wave (CW) and Pulsed. The CW mode is associated with communication transmitting devices such as radio relay and television. The Pulsed mode is associated with radar, and industrial and medical equipment. From a biologic standpoint, the Pulsed mode is considered to be the more significant, due primarily to the high power intensities and energy distribution.

Microwave energy, because it is frequently referred to as microwave radiation, is often confused with ionizing radiation. This is unfortunate since there are no important similarities between the two energies so far as biologic effects are concerned.

Microwave radiation is thought to be similar to infrared radiation in that it causes localized heating of the skin, but penetration is deeper. There is laboratory evidence based on direction of stationary pulsed beams at animals, that microwave radiation can cook underlying muscle, produce cataracts, and cause death by hyperthermia. There have been a few reports of harmful human effects such as transient superficial heating of the skin, heating of a steel fracture plate, and development of cataracts, but there have been no proved reports of more serious human injury or death from radar exposure.

The main reasons for the observed response in man include—

(1) The human body has an efficient heat regulating system capable of resisting microwave heat better than laboratory animals.

(2) The larger field intensities of microwave energy are associated with radar transmitters whose antennas are mounted on towers, either as stationary objects aimed at a point near the horizon or as rotating or rocking objects aimed both below and above the horizon. In the case of stationary antennas, the concentrated beam of energy cannot normally searchlight unprotected humans on the ground. In the case of rotating or rocking antennas, any searchlighting is of an intermittent nature, a situation which provides the worker an opportunity to lose heat during nonexposure periods.

(3) Work is generally performed in open areas making possible the loss of body heat to the cooler surrounding air.

(4) Existence of adequate health and safety programs to minimize the hazards of microwave radiation.

It is not to be inferred that the problems associated with microwave hazards require only minimum attention. Indeed, caution should be observed and specific controls adopted as dictated by the type of radar set used. The possibility of the introduction in the near future of more powerful radar units introduces additional concern. The lack of conclusive information on the existence or nonexistence of nonthermal effects might be accounted for by the absence of appropriate detection techniques.

There is no generally acceptable maximum safe exposure level, but an average power density of 0.01 watt per square centimeter for all frequencies has been suggested as an arbitrary maximum ambient power level. It should be borne in mind that high field strengths can be associated with low average power. Even though no field-strength dependent effects have been demonstrated, the possibility of such effects should not be overlooked, and caution should be exercised by those working in pulsed fields of high peak, but low average, power.

*Potential occupational exposures*—Occupations potentially associated with microwave radiation exposures include the following.

Air crewmen  
Chemists

Drug sterilizers  
Food sterilizers

Furniture veneering operators	Microwave testers
Maser operators (microwave amplification by stimulated emission of radiation)	Missile launchers
Microtherm operators	Radar mechanics
Microwave development workers	Radar operators
Microwave diathermy operators	Radio frequency oven maintenance workers
	Radio frequency oven operators

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## (2) Ionizing Radiation

Man has always been exposed to ionizing radiation. Thus the nature of the hazard itself is not new. The change that has occurred in recent times is in the size of the hazard. Ionizing radiation refers to (1) electrically charged or neutral particles, or (2) electromagnetic radiation which will interact with gases, liquids, or solids to produce ions. A large number of such particles are known to nuclear physicists, most of which will not usually be important factors in occupational exposures to ionizing radiation.

Depending upon their physical characteristics, the numerous kinds of ionizing radiation may be classified either under the corpuscular (or particulate) radiations or the electromagnetic wave type of radiation. The first type contains particles such as alpha particles, beta particles, neutrons, and protons; these particles have mass and are electrically charged with the exception of the neutron, which carries no charge. The second, or electromagnetic wave type, has associated with it uncharged electromagnetic vibrations such as gamma rays and X-rays.

The several types of ionizing radiation determined by these different particles and rays vary in their penetrative powers, and also with regard to the number of ions left in their tracks as they move through tissue. Biologic effect varies, among other factors, with ion density; that is to say, with the number of ions produced per unit length of track.

Ionizing radiations are produced naturally by radioactive decay processes or artificially by such devices as high energy accelerators. A radioactive nucleus is one that spontaneously changes to a lower energy state, emitting particles and gamma rays in the process. The particles commonly emitted are alpha particles and beta particles. High energy accelerators can produce all of the above particles and, additionally, protons and X-rays.

### *Alpha Particles*

An alpha particle is the nucleus of a helium atom. It is a heavy particle with a weight of four mass units, and since it consists of two protons, two neutrons, and no electrons, it carries two positive charges. Alpha particles interact readily with matter to produce ions and usually have energies of from 4 to 8 million electron volts (Mev). They will travel a few centimeters in air and up to 60 microns in tissue. Their high energy and short path mean that they produce a dense track of ionization along their path and thus can produce serious biologic damage in the tissues with which they interact. Alpha particles will not penetrate the cornified layer of the skin and thus are not an external hazard. However, if alpha-emitting elements are taken into

the body, they create serious exposure problems. Examples of such elements are radium, radiothorium, and polonium.

### *Beta Particles*

A beta particle is either a negatively charged electron or a positively charged positron, created by nuclear processes in which a neutron changes to a proton and an electron, or a proton changes to a neutron and a positron. Beta particles produced by radioactive decay have energies ranging from essentially zero to a definite maximum which is characteristic for each element. One of the most energetic naturally produced beta particles ( $E_{\max} = 3.1$  Mev) occurs in the RaC to RaC'' transition. Artificial beta particles produced by accelerators such as Van de Graaff machines or betatrons may have energies up to 100 Mev.

Beta particles interact much less readily with matter than alpha particles and will travel up to a few centimeters in tissue or many meters in air. Exposure to external sources of beta particles is potentially hazardous, and beta-emitting elements which are taken into the body create internal hazards.

### *Protons*

A proton is the nucleus of a hydrogen atom and is relatively heavy with a single positive charge. Protons are produced by high-energy accelerators usually with energies of a few Mev. They are quite effective in producing tissue ionization, and their path length is somewhat longer than the path of alpha particles of equivalent energy.

### *Gamma Rays and X-rays*

Gamma rays and X-rays are electromagnetic radiations with similar properties. X-rays, in general, have longer wave lengths, lower frequencies and therefore lower energies, than gamma rays. Gamma rays are produced by nuclear processes, while X-rays result from the interaction of high-speed electrons with the atoms. Naturally produced gamma rays have energies ranging from a few kiloelectron volts (Kev) to a few Mev, while high-energy accelerators can produce gamma rays of a few hundred Mev. X-ray energies form a continuous spectrum from the very low energies (or frequencies) in the upper ultraviolet range up to the energies of the high speed electrons that are producing the X-rays, often about 100 Kev. Gamma rays and highly penetrating X-rays produce a low ion density in the matter with which they interact. They are primarily an external hazard, and their biologic effects are better known than those of any of the other ionizing radiations. Examples of gamma emitters used in industry are cobalt-60 and iridium-192. In addition to being generated by X-ray tubes, X-rays may be encountered during the manufacture and use of klystron tubes and electron microscopes.

## *Neutrons*

Neutrons are uncharged nuclear particles, which, together with protons, compose the nuclei of all elements except hydrogen-1. They have approximately the same mass as a proton, but since they are electrically neutral they react with matter in a different manner. Neutrons lose energy by direct collisions with nuclei or by entering a nucleus and initiating a nuclear reaction. The biologic effects of neutrons are primarily caused by charged particles and secondarily emitted gamma rays produced by these collisions or reactions.

Neutrons are produced by nuclear reactors or from sources such as radium-beryllium or polonium-beryllium mixtures. They also are produced by high-energy accelerators such as cyclotrons, with energies ranging from thermal (0.03 electron volt) to several Mev. The relative biologic effectiveness of neutrons is dependent on the energies of the neutrons.

## *Biologic Effects*

The fact that ionizing radiation can cause biologic damage has been well documented over a period of years, and limits for occupational exposure to external radiation and for concentrations of radioactive isotopes in air and in water have been recommended by the National Committee on Radiation Protection and published in National Bureau of Standards Handbooks 59 and 69. For external radiation, the data are based on records of human exposure. For internal exposure, sufficient records are available for radium only. The values for other bone-seeking elements have been assigned by comparison with radium and from the results of animal experiments. For most of the other isotopes the exposure limit values were calculated, using the concept that the dose to the critical organ should not exceed 100 millirems per week.

Methods of preventing over-exposure to either external or internal radiation have been developed and should be followed carefully to prevent damage to the workers. Pertinent material may be found in National Bureau of Standards handbooks listed in Handbook 69.

The biologic effects of ionizing radiation may be divided into the somatic and genetic, the somatic including such effects as acute and chronic radiodermatitis, acute and chronic radiation syndrome, skin cancer, leukemia, cataracts, sterility, and shortening of life span. The genetic effects resulting from occupational exposures are unknown. Moreover, a mutation produced by radiation is probably similar to one effected by a mutagenic chemical or to one occurring spontaneously.

The effects of occupational exposure to ionizing radiation are usually localized, with production of acute or chronic radiodermatitis. Generalized exposure to penetrating ionizing radiation, and the resulting acute radiation syndrome, are rare in industry and are usually associated with an accident. Chronic occupational exposure to low levels of ionizing radiation is also rare

but may produce leukopenia and anemia. Other rare effects from occupational exposure to ionizing radiation include cataracts among cyclotron operators, bone sarcoma among radium dial painters, and cancer of the lung among pitchblende miners.

A commonly recognized effect of ionizing radiation is acute or chronic radiodermatitis. Mild acute exposures are most common in the applications of radiotherapy, while chronic exposures are most common in industry. Acute radiodermatitis, regardless of the type of ionizing radiation involved, presents the same clinical appearance, which is graded into three degrees of damage to the skin and its appendages, thus:

*1st degree:* Erythema after several hours to several days, followed by hyperpigmentation and temporary alopecia.

*2nd degree:* Erythema and edema followed by vesicles or bullae formation, superficial ulceration, permanent alopecia, permanent loss of nails and glands (sweat glands may regenerate), and atrophic, telangiectatic scarring.

*3rd degree:* Erythema and deep edema, followed by necrosis and slough; healing is slow and difficult or never occurs, leaving an ulcer; underlying tissues such as cartilage and bone may be involved.

Chronic radiodermatitis results from third degree radiodermatitis or from repeated suberythema exposures. The cutaneous effects of ionizing radiation, like those of ultraviolet radiation, are cumulative. Grossly, four clinical effects are seen in chronic radiodermatitis: atrophy, telangiectases, hypopigmentation, and hyperpigmentation. If the fingers are involved, and they frequently are, the skin markings of the finger tips may lose their ridges, but this may also be seen in workers handling heavy and rough metal parts, as well as in pottery workers. The finger nails may become fragile and show longitudinal striations. Eventually, there may develop ulcerations, senile keratoses, and squamous cell epitheliomata. Basal cell epitheliomata can also develop in areas of chronic radiodermatitis, though less commonly than the squamous cell type.

Accidental whole body radiation with doses greater than 100 roentgens usually results in the acute radiation syndrome. Initial symptoms are nausea, vomiting, weakness, and shock. Death during this stage has been called a central nervous system death. Following a latent period of two days to two weeks, later symptoms begin with malaise and fever. There are hemorrhagic lesions of the skin, and by the third week epilation occurs. Painful ulcerations occur in the mouth and throat, and simultaneous ulcerations in the gastrointestinal tract produce bloody diarrhea. By this time anemia, leukopenia, and thrombocytopenia appear. Death may result from the bloody diarrhea or from severe bone marrow depression.

## *Early Recognition of Exposure*

Several tests for the early recognition of the effects of ionizing radiation have been described. These are based on certain hematologic findings, such as leukopenia, relative lymphocytosis, presence of abnormal monocytes, increased incidence of bilobed lymphocytes, and an increase of deoxyribonucleic acid in lymphocytes. While these tests may be helpful in certain instances, the findings are not specific and should not be accepted in themselves as evidence of exposure to ionizing radiation. Nor should they be accepted as substitutes for personal and environmental monitoring devices.

## *Industrial Uses*

Each year additional industrial and medical applications of ionizing radiation are discovered. Industrial uses include the following.

Abrasion measurements	Gelatin production
Atomic battery fuel	Graft polymerization of plastics
Automation	Hardening of plastics
Blast furnace study	Instrument calibration
Blight control	Interface labelling of liquids
Byproduct power	flowing through a pipeline
Catalytic and other application to solids	Land propulsion
Chemical processing	Leakage rate measurements
Coke oven operation	Leak detection in buried pipeline
Crank shaft inspection	Liquid level gages
Crop storage	Location of obstruction in pneumatic tubes
Density gages, cigarette	Location of oil holes and other outlets in metals and other dense products
Detection of gasket leaks and other defects	Lubricating oil production
Detection of voids, and defects in welds, forgings, castings and other solids	Luminous compound manufacture
Determination of corrosion in high pressure steam lines, gas mains and acid tanks	Measurement and control of thickness in rolling steel, linoleum and other products
Determination of dustiness	Oil well logging
Determination of effectiveness of cleansers and cleansing machines	Oil well stimulation
Disinfestation of products	Ore assaying
Distillation of sea water	Preservation of foods
Ethyl bromide production	Prevention of potato sprouting
Fire alarm making	Process heat
	Regulation and measurement of flow of liquids and solids

Safety controls  
 Silicone rubber vulcanization  
 Snow depth measurements  
 Space heat  
 Static elimination  
 Sterilization of drugs  
 Sterilization of medical supplies

Sulfurimeters  
 Thickness gages in rolling steel  
 and in the production of metals,  
 rubber and tire fabric, plastics  
 and adhesives, and paper  
 Vacuum gages

### *Potential Occupational Exposures*

With the widespread use of radioactive isotopes in industry and the increasing use of X-ray sources, radiation exposures may occur in a wide variety of occupations. In addition to apparent sources such as thickness gages and radiographic equipment, incidental sources occur in industry; examples are klystron tubes and radar tube-testing operations. In general, any operation where a high voltage electron beam impinges on metal will incidentally create X-rays and should be shielded.

While users of Atomic Energy Commission-produced isotopes and source material are licensed by that agency, there are no such restrictions on cyclotron-produced materials or on natural elements such as radium, polonium, and radiothorium. Such materials, therefore, might be used by groups of widely varying competence. Several States require the registration of radiation sources, but some do not. It is pertinent to note in connection with the addition in 1959 of Section 274, *Cooperation with States*, to the Atomic Energy Act of 1954 that the Atomic Energy Commission is placing emphasis on the desirability of the States assuming some of the Commission's regulatory authority. Such authority, when accepted, would enable the State to control by-product material, source material, and special nuclear material in quantities not sufficient to form a critical mass. Kentucky was the first State to accept authority, the agreement becoming effective March 26, 1962.

The following examples show the diversity of occupations potentially exposed to ionizing radiation.

Aircraft workers  
 Atomic energy plant workers  
 Biologists  
 Cathode ray tube makers  
 Ceramic workers  
 Chemists  
 Dental assistants  
 Dentists  
 Dermatologists  
 Drug makers  
 Drug sterilizers  
 Electron microscope makers

Electron microscopists  
 Electrostatic eliminator operators  
 Embalmers  
 Fire alarm makers  
 Food preservers  
 Food sterilizers  
 Gas mantle makers  
 Glass makers  
 High voltage television repairmen  
 High voltage vacuum tube makers  
 High voltage vacuum tube users  
 Industrial fluoroscope operators

Industrial radiographers	Prospectors
Inspectors using, and workers in proximity to, sealed gamma ray sources (cesium-137, cobalt-60 and iridium-192)	Radar tube makers
Klystron tube operators	Radiologists
Laboratory technicians	Radium laboratory workers
Liquid level gage operators	Radium refinery workers
Luminous dial painters	Research workers
Machinists, fabricated metal product	Roentgenologists
Oil well loggers	Roentgen tube makers
Ore assayers	Shoe fitters
Pathologists	Television tube makers
Petroleum refinery workers	Thickness gage operators
Physicians	Thorium-aluminum alloy workers
Physicists	Thorium-magnesium alloy workers
Pipeline oil flow testers	Thorium ore producers
Pipeline weld radiographers	Tile glaziers
Plasma torch operators	Uranium dye workers
Plastic technicians	Uranium mill workers
Pressman, printing	Uranium miners
	Veterinarians
	X-ray aides
	X-ray diffraction apparatus operators
	X-ray technicians

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### (3) Abnormal Air Pressure

Abnormal air pressure may be considered to be any pressure either above or below normal sea level pressure of 14.7 pounds per square inch. The normal of 14.7 pounds is equivalent to 1 atmosphere. Potential occupational exposure to more than 1 atmosphere of compressed air is associated with those who work in caissons or in tunneling operations where a compressed air environment is utilized to exclude water and to aid in supporting the structure. Potential occupational exposure is also associated with those

who dive into the depths of the sea, whether they breathe from a self-contained underwater breathing apparatus (SCUBA) or from a hose bringing air from a surface compressor. Pressure below 1 atmosphere is a potential hazard of pilots and crews of high performance aircraft in flight.

### *Increased Air Pressure*

It has long been recognized that exposure to a compressed air environment with subsequent return to normal ambient air pressure levels may result in the production of signs and symptoms of alarming severity.

An increasingly large number of physicians throughout the country are being faced with the problems of recognizing, preventing, and treating the many consequences of compressed air exposure. This fact is due largely to the activities of two groups; namely, the skin divers using SCUBA and the tunnel builders, who because of expanding urban development must burrow deeper and longer to complete the necessary sewer and aqueduct construction.

It is well known that excessively rapid decompression of divers and compressed air workers gives rise to the formation of nitrogen bubbles in the blood stream and body tissues, and that these bubbles, if they are of sufficient size and number and if they are formed in or carried to critical areas, may give rise to one or more of the many signs and symptoms of compressed air illness or decompression sickness.

While the hazards of rapid decompression are great, one must also be mindful of the untoward effects produced by exposure to compressed air itself.

The effects of compression, that is, the effects of increasing air pressure on the body, will be considered first, followed by a discussion of the effects of decompression or the effects of returning the body to normal or sea level air pressure.

### *Primary Pressure Phenomena*

The body can be compressed to almost 18 atmospheres without demonstrable changes attributable to the compression itself provided that air has free access to all surfaces of the body including the sinuses and middle ear spaces. If, however, the pressure is not equally distributed over all body surfaces, a pressure difference between tissues and the ambient atmosphere of less than 1 pound per square inch will produce congestion, edema, hemorrhage, and pain in the tissues exposed.

The ears, sinuses, and teeth are common sites of injury resulting from *squeeze* due to increased air pressure. About 1.5 percent of exposed individuals are subject to dental pain or barodontalgia and another 1.5 percent are subject to sinus pain or barosinusitis. Dental pain suggests the presence of small gas bubbles in the pulp or in a part of the tooth where soft tissues can be squeezed. Sinus pain is probably due to occlusion of the sinus aperture by inflamed mucous membrane or lymphatic tissue thus preventing

the requisite air from entering or leaving the sinus to effect an equalization of internal and external pressures.

If the eustachian tube is blocked by lymphoid tissue or swollen mucous membrane, a pressure difference will be created between the middle ear and the external environment. As this pressure gradient increases and as the duration of exposure is prolonged, certain progressive changes occur in the ear drum and in the middle ear. These changes, referred to as barotitis, begin with congestion of the ear drum and progress through erythema, and contraction of the drum and hemorrhage into it. This may be followed by hemorrhage into the middle ear itself. However, properly treated by expectancy, this condition will resolve itself and no sequelae will occur.

The lungs themselves may be subject to squeeze if the chest is compressed to a volume smaller than the amount of residual air of the lung, normally the amount of air left in the lungs following forced expiration. *Lung squeeze* is occasionally seen in unprotected swimmers who dive by simply holding their breath. The effect of the squeeze is to force blood and tissue fluids into the respiratory passages and alveoli. Considerable lung damage may result.

### *Secondary Pressure Phenomena*

*Narcotic action of nitrogen*—The phenomena previously described are effected primarily by the formation of a pressure gradient between a body cavity and the external environment. On an entirely different basis are those pressure phenomena associated with disturbances in gaseous equilibria. At 4 atmospheres of pressure or more, the gaseous nitrogen induces a narcotic action evidenced by decreased ability to work, mood changes, and frequently, a mild to marked euphoria. The responses are, in fact, similar to those associated with alcoholic intoxication. The exact cause of this cerebral disturbance is unknown. However, it may be noted that nitrogen is highly soluble in fat, the ratio of its solubility in fat to its solubility in water being about 5 to 1. According to the Meyer-Overton hypothesis, a gas having such a relatively high ratio may act as a narcotic.

*Oxygen poisoning*—Inhalation of oxygen when its partial pressure exceeds 2 atmospheres, or about 30 pounds per square inch (gage) may result in the production of the signs and symptoms of oxygen poisoning.

The mechanism of oxygen toxicity is not well understood. The signs and symptoms of this intoxication may include tingling of fingers and toes, visual disturbances, acoustic hallucinations, confusion, muscle twitching, especially about the face, nausea, and vertigo. The final result of such exposure may be the epileptiform convulsion, which ceases as soon as exposure to high oxygen partial pressures is terminated. This toxic action of oxygen is greatly enhanced by exercise or by the presence of moderate amounts of carbon dioxide.

At normal atmospheric pressures, pure oxygen will irritate the throat and nasal membranes after about 12 hours of breathing time but signs and symptoms of systemic oxygen poisoning do not occur.

It should be noted that the greatest hazard in oxygen administration in chambers is the danger of fire.

*Effect of carbon dioxide*—Carbon dioxide enhances the toxicity of oxygen and the narcotic effect of nitrogen, and in addition a higher incidence of bends has been reported in association with a rise in the CO<sub>2</sub> level. The concentration of CO<sub>2</sub> present in the breathing media in a compressed air environment should not exceed the equivalent of 0.2 percent of one atmosphere.

### *Effects of Decompression*

During ascent from the depths or during decompression in a chamber, two major groups of problems are introduced. The first group is a result of the property which allows a gas to expand as pressure decreases. The second is due to the tendency for dissolved nitrogen to escape from solution in the form of bubbles as pressure decreases.

The most serious effects of decompression are produced by the expansion of air in the lungs. If air is taken into the lung at a depth of 125 feet (55 pounds per square inch, gage), it will increase in volume five times when decompression to atmospheric pressure occurs. If decompression is excessively rapid and sufficient air is not exhaled, some of the pulmonary alveoli will rupture with the formation of one or more of the following: mediastinal emphysema, pneumothorax, or air embolism. The most dangerous of these conditions is the air embolism which occurs when air, expanding in the lung, is forced into the pulmonary blood vessels and then into the left side of the heart and into the arterial circulation which may quickly carry the air bubbles to the brain and produce a cerebral air embolism, a condition which may be rapidly fatal if not treated promptly by recompression.

If a given exposure to high pressure has caused a sufficient amount of nitrogen to be dissolved in the blood and in the tissues, and if decompression occurs sufficiently fast, gas bubbles will be formed. These bubbles of liberated gas create a condition of circulatory impairment and local tissue destruction which in turn are responsible for the signs and symptoms of decompression sickness.

The amount of bubble formation that will occur upon decompression depends to a large extent upon the following three factors: (1) the amount of gas dissolved in the tissues, which in turn is dependent upon the degree and duration of exposure to pressure and upon the amount of body fat in which the gas can be dissolved; (2) conditions which alter blood flow, including age, temperature, exercise, fright, and post-alcoholic state, especially if these alteration in blood flow occur during or shortly after the decompression.

sion process; and (3) the rapidity of decompression from elevated air pressure to the ambient level.

### *Nitrogen Bubble Formation: Signs and Symptoms*

*Bends*—A relatively common manifestation of compressed air illness is described by Behnke as being a dull, throbbing type of pain which is gradual in onset, progressive and shifting in character, and frequently felt in the joints or deep in the muscles and bones. When the symptoms of bends occur, they do so in the first 4 to 6 hours in 80 percent of the cases, while the remainder will occur within 24 hours. Contributing to variations in susceptibility are such factors as age, obesity, defects of the lungs, heart impairments, temporary ill health, and individual predisposition.

*Chokes*—This rather specific type of asphyxia occurs less frequently than bends and is thought to be due to the accumulation in the large veins, the right side of the heart, and the pulmonary vessels of quantities of gas eliminated from the arterial circulation and from the extravascular tissues. The earliest evidence of impending chokes is a sensation of substernal distress felt during deep inspiration, especially during inhalation of tobacco smoke, which elicits paroxysmal coughing (Behnke's Sign). These attacks of coughing may proceed to loss of consciousness with all of the signs and symptoms of a true shock-like syndrome.

*Paralysis*—The most serious complication of decompression sickness is paralysis. Spastic paraplegia or monoplegia involving the lower extremities may follow improper decompression resulting in the formation of bubbles in the blood vessels and tissues of the spinal cord. Immediate and prolonged recompression usually brings about rapid recovery even following paraplegia. Cerebral involvement is very rare.

*Aseptic bone necrosis*—One important sequela of compressed air exposure is the development of aseptic bone necrosis. This condition is thought to be caused by the occlusion of small arteries in the bone by bubbles of nitrogen followed by infarction of the involved area. The sites of predilection for the occurrence of occlusion and necrosis, as seen in this process, are the lower femoral diaphysis, the upper tibial diaphysis, and the head and neck of the humerus and the femur. These lesions are usually multiple and tend to be bilaterally symmetrical.

Aseptic bone necrosis is usually asymptomatic unless joint surfaces are involved, in which case pain may be a symptom. Complete collapse of the affected joint has been known to occur. Healing takes place through an osteocondensing process. This increase in density may appear on roentgenographic examination as a *snowcap* on the top of the articular surface.

It is thought that prolonged daily exposure for at least 8 months is necessary to produce this type of bone necrosis since it is seen in tunnel workers but seldom in divers.

It is probable that the occurrence of aseptic bone necrosis can be avoided if strict observance of recommended decompression schedules is adhered to.

### *Decreased Air Pressure*

When referring to the symptom complex developing as a result of exposure to high altitudes, *dysbarism* is the preferred term. That of decompression sickness is perfectly acceptable and includes the entire symptom complex which may develop. However, as determined by common usage, this term more often refers to the syndrome that develops in deep sea divers upon their return to the surface following exposure to several atmospheres of pressure.

The signs and symptoms of dysbarism result from the expansion of the gases within the body cavities and from the formation of nitrogen bubbles in body tissues and fluids from gas which is normally in solution at sea level pressure. Upon traveling to high altitudes, the resultant reduction in barometric pressure allows the gases within the body to expand to a greater volume than occupied at sea level. One volume of gas at sea level becomes two volumes at 18,000 feet, three at 28,000 feet, four at 33,000 feet, and five at 38,000 feet. On descent the volume changes in reverse order.

The greatest hazard at altitude is lack of oxygen; however, bends, chokes, neurologic disorders, aeroembolism, aerodontalgia, aerotitis, and aerosinusitis may all be experienced by those exposed to an environment of significantly decreased air pressure, that is, one above 30,000 feet.

Dysbarism may be complicated by a type of neurogenic peripheral circulatory failure or primary decompression shock consisting of any or all of the following manifestations: intense pallor, profuse sweating, faintness and dizziness, nausea, vomiting, loss of consciousness. These symptoms are usually rapidly relieved by descent from altitude.

The syndrome of primary decompression shock is much more commonly encountered following exposure to altitude than to depth. Spinal cord involvement is rarely a sequela of dysbarism but is not uncommon in divers. Aseptic bone necrosis is essentially unknown in pilots; is rare in divers; but is common in caisson and pressurized tunnel workers.

### *Potential Occupational Exposures*

Occupations potentially associated with abnormal air pressures include the following.

#### *Increased Air Pressure*

Caisson workers	Mine tunnelers
Canal tunnelers	Railroad tunnelers
Divers	Road tunnelers
Drain tunnelers	Sewer tunnelers

#### *Decreased Air Pressure*

Air crewmen	Airplane pilots
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### (4) Abnormal Temperature

#### Heat

There are four distinct factors which influence the interchange of heat between man and his environment. These factors are the following.

- (1) Air temperature
- (2) Air velocity
- (3) Moisture content of the air
- (4) Radiant temperature

The industrial heat problem is one in which a combination of these factors interacts to produce a working environment which may be uncomfortable or even hazardous to the worker concerned.

The fundamental thermodynamic processes involved in heat exchange between the body and its environment may be described by the basic equation of heat balance. This equation reads as follows.

$$M = \pm S - E \pm R \pm C \quad (1)$$

where M=rate of metabolism; S=rate of storage, or change in body heat content; E=rate of heat loss through evaporation; R=rate of heat loss or gain by radiation; and C=rate of heat loss or gain through convection.

Under conditions of comfort, this balance can be expressed as shown in 2.

$$M - (\pm S - E \pm R \pm C) = 0 \quad (2)$$

For purposes of temperature determination, the body can be divided into two regions, the deep region or the *core*, and the superficial region, which is made up largely of the skin and subcutaneous tissues.

The heat regulatory mechanisms of the body are directed at keeping the core temperature at a uniform level, while the temperature of the superficial tissues may vary within a relatively wide range according to the amount of heat received from the environment.

When heat loss fails to keep pace with heat gain, the core temperature begins to rise and certain physiologic mechanisms come into play in an attempt to increase heat loss from the body. First, there is a dilation of the blood vessels of the skin and subcutaneous tissues with a diversion of a large part of cardiac output to these superficial regions. There is a concomitant increase in circulating blood volume brought about by contraction of the spleen and by dilution of the circulating blood with fluids drawn from other tissues. Cardiac output is also increased. It is probable that neither thyroid nor adrenal hormones play a part in increasing body heat loss.

In equation 1,  $M \pm R \pm C$  may be taken to indicate the total heat load imposed by the combined effects of metabolic and environmental heat. The evaporative capacity required to maintain body heat balance,  $E_{req}$ , is then equal to  $M \pm R \pm C$ . If  $E_{req}$  exceeds the maximum evaporative capacity of the body,  $E_{max}$ , a condition of stress will develop. Many heat stress indexes have been constructed, but each contains inherent limitations because of the conditions, usually experimental, under which they have been developed.

Acclimatization is essential if man is to withstand prolonged increased heat loads. This process of adaptation is characterized by the worker's ability to perform with less increase in core temperature and by the secretion of decreased amounts of perspiration. This perspiration is more dilute, that is, it contains a lower concentration of sodium chloride than the perspiration of a nonacclimatized individual. Thus essential salt is thereby conserved. Acclimatization to heat occurs in from 1 to 2 weeks.

In general, industrial heat exposures may be classified as either hot-dry or as warm-moist. Hot-dry environments are found in industrial situations in which ambient and radiant temperatures are elevated but in which moisture content of the air is not excessive. The difficulties in hot-dry situations arise when the body absorbs more heat by radiation or convection or both than it can lose through evaporation of perspiration, that is,  $R \pm C$  exceeds  $E_{max}$ . Warm-moist working environments may be encountered in occupations where large amounts of moisture are released from the industrial processes involved, but where ambient and radiant temperatures are only moderate. Here, the heat load from radiation or convection, or both, is not great, but the high moisture content of the air inhibits heat loss from the body through the

perspiration mechanism. In such an environment,  $E_{max}$  is diminished because of the moisture content of the surrounding air.

### *Psychologic Reactions*

Psychologic reactions to prolonged exposure to excessive heat include increased irritability, lassitude, decrease in morale, increased anxiety, and inability to concentrate. The results are mirrored by a general decrease in the efficiency of production and in the quality of the finished product.

### *Physical Reactions*

Physical reactions to prolonged exposure to excessive heat include heat cramps, heat exhaustion, and heat stroke.

*Heat cramps*—These cramps may occur after prolonged exposure to heat attended by profuse perspiration with resultant loss of large amounts of salt. The signs and symptoms of heat cramps consist of spasm and pain in the muscles of the abdomen and extremities. Albuminuria may be a transient finding.

*Heat exhaustion*—This condition may result from physical exertion in a hot environment when vasomotor control and cardiac output are inadequate to meet the increased demand placed upon them by peripheral vasodilatation. Signs and symptoms of heat exhaustion may include lassitude, dizziness, syncope, profuse sweating, and cool moist skin. There is usually no attendant hyperthermia.

*Heat stroke (sunstroke)*—This is a much more serious condition than heat cramps or heat exhaustion. An important predisposing factor is excessive physical exertion. Heat pyrexia is closely related to humidity as well as to temperature and is less common in areas where relative humidity is very low. Signs and symptoms may include dizziness, nausea, severe headache, hot dry skin, very high body temperature (often greater than 108° F.), coma, and death.

### *Potential Occupational Exposures*

An attempt to compile a list of occupations potentially associated with hot environments would probably result in a list of *hot jobs* having little usefulness. Climatic conditions and plant layout are probably factors of greater significance than heat per se; thus heat stress may be involved in many operations not categorized as hot jobs. Of importance, rather, is the recognition of the signs and symptoms previously referred to, which, in turn, will suggest appropriate treatment and environmental control. Finally, in the evaluation of an environmental heat problem, it is helpful to remember that some physiologic responses may be associated with ambient temperatures at levels much lower than generally believed, and that the age of the worker may be a factor of some importance in the physiologic reactions produced by the job.

## Cold

As the environmental temperature is reduced below normal body temperature, equation 1 may be rearranged, as shown in 3, to express the change in heat equilibrium that may occur.

$$S = M - E - R - C \quad (3)$$

The change in body heat content is thus equal to the heat gain through metabolism minus the loss of heat through evaporation, radiation, and convection. It is evident that the body will tend to lose heat to the environment if heat loss by evaporation, radiation, and convection exceeds body heat gain from metabolic processes. In general, cooling stress is proportional to the total gradient between the skin and the environmental temperature since this gradient determines the rate of heat loss from the body by radiation and convection. Loss of heat through the mechanism of the evaporation of perspiration is not significant at temperatures below normal body temperatures.

For the body to maintain thermal homeostasis in a cold environment, certain physiologic mechanisms come into play which tend to limit heat loss and increase heat production. The first mechanism is one of peripheral vasoconstriction, especially in the extremities, resulting in a marked drop in skin temperature. Body heat loss to the environment is thereby diminished.

When vasoconstriction is no longer adequate to maintain body heat balance, muscular hypertonus and shivering become important mechanisms for increasing body temperature by causing metabolic heat production to increase to several times the normal rate. Final breakdown of thermal control depends upon the degree of physical activity of the worker; amount of clothing worn; and the nature, intensity, and duration of exposure to the environment. In the event of such a breakdown, the following may occur.

(1) If activity is restricted, the extremities, notably toes and fingers, approach freezing temperatures most rapidly. A depression of general body temperature follows. (2) If the individual is physically active, cooling develops with fatigue, and as exhaustion approaches, the vasoconstrictor mechanism is overpowered and sudden vasodilatation occurs with resultant rapid loss of heat. Critical cooling then ensues.

## Physical Reactions

In addition to the above-mentioned generalized physiologic reactions to cold stress, certain localized phenomena may occur. These include trench foot and frostbite.

*Trench foot*—If there is long continuous exposure to cold without freezing combined with persistent dampness or actual immersion in water, a syndrome referred to as trench foot or immersion foot may be produced. This condition is due to persistent local tissue anoxia, combined with mild or severe cold with resultant injury to the capillary wall. Edema, tingling, itching, and

severe pain occur and may be followed by blistering, superficial skin necrosis and ulceration.

*Frostbite*—Frostbite, in contradistinction to trench foot, occurs when there is actual freezing of the tissues with the attendant mechanical disruption of cell structure. Frostbite results from exposure to cold air, usually at temperatures below 10° F. Theoretically, the freezing point of the skin is about 30° F., but, when dry and oily, the skin's property of supercooling without freezing affords considerable protection. Once started, however, freezing progresses rapidly. The tissues of the cheeks, nose, ears, and digits are the first to be injured. The first warning of frostbite is often a sharp, pricking sensation. However, cold itself produces numbness and anesthesia which may permit serious freezing to develop without the warning of acute discomfort. Injury produced by frostbite may range from simple redness of the skin with transient anesthesia and superficial bullae to persisting ischemia, thrombosis, deep cyanosis and gangrene.

### *Abnormal Responses to Cold*

Certain vascular abnormalities may be either precipitated or aggravated by cold exposures. These include chilblain, pernio, Raynaud's disease, acrocyanosis, and thromboangiitis obliterans. Workers suffering from these ailments should take special precautions to protect against chilling.

### *Potential Occupational Exposures*

Occupations potentially associated with cold environments include the following.

Arctic research technicians	Linemen
Bridge builders	Liquefied gas workers
Bridge tenders	Loggers
Construction workers	Mail carriers
Divers	Packing-house workers
Dock workers	Pipeline workers
Dry ice workers	Policemen
Farmers	Refrigeration workers
Firemen	Road builders
Fishermen	Salvage workers
Harbor workers	Shepherds
Highway workers	Surveyors
Ice makers	Tunnel traffic workers

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## (5) Defective Illumination

The visibility of an object is determined by four factors. These are size, contrast, time of viewing, and brightness. As the size of the object being viewed increases, visibility increases and, up to a certain point, seeing becomes easier. Within limits, higher contrast between the object being viewed and its background means greater visibility of that object. Time of viewing is equally important in that lower levels of illumination require an increased amount of time of viewing to accomplish a visual task. Within certain limits, increased brightness will usually mean increased visibility.

In most tasks the factors of size and time of viewing are constant or hold little flexibility. Contrast, by the same token, may also be limited to a great extent by the nature of the task itself. Therefore, brightness is in general the most important controllable factor in the visibility of an object.

The effects of defective illumination are legion. A large share of all industrial accidents may be traced to this issue. Also, many specific as well as general effects may be seen in workers subjected to this hazard. Defective illumination may cause a feeling of general discomfort, muscular tension, eye fatigue, increased blinking of the eyes, a decrease in convergence reserve, a decrease in visual acuity, aggravation of eye defects, dizziness, headache, and even blindness. The worker may show the effects of this hazard in a more general, subtle way, such as a decrease in his efficiency and work output, increase in errors in his work, and a generally low morale.

We can all attest to the observation that our *spirits* or *emotions* tend to be low or high according to the brightness of our surroundings. Alertness, cheerfulness, and increased performance are all favored by the presence

of good illumination, while a general feeling of dullness and depression is frequently associated with the drab environment of a poorly lighted room.

Defective or unsuitable illumination will result when an abnormal quantity of illumination is used, either too low or too high. It may also be produced by glare, unsuitability of color of light source or surroundings, and shadows. Any one, or a combination of the factors producing defective illumination may be present in almost any working situation. The importance of each varies with the particular type of industry.

### *Low Illumination*

Miners' nystagmus, evidenced by the rhythmic involuntary movement of the eyes, has been attributed to continued use of the eyes under conditions of very low illumination. Miners afflicted with this disease may become incapacitated for work. The disease is becoming less of a problem in Great Britain and continues to be rare among miners in the United States. The continued infrequent occurrence of the disease among American miners has been attributed to the level of illumination in the mines.

Data from Great Britain on the hazards of coal mining illustrate the part played by illumination. The gradual improvement of underground illumination was accompanied by a decrease in the number of coal miners certified as having the disease. Thus, during the years immediately prior to World War II, the annual certification rate was about 150 per 100,000 miners. In 1950 the corresponding rate was 34, and by 1960 it had decreased to 9. It would appear that, while discussions of miners' nystagmus frequently point to the psychologic factor as being responsible for the disease, illumination, regardless of its place in the chain of causation, cannot be ignored.

Miners' nystagmus may be seen in a latent, or subacute form, and in an acute form. Symptoms are first a marked loss of visual acuity, especially at night, oscillation of the eyes when fatigued, photophobia, and giddiness associated with bending. In the more acute forms, headache may be a complaint, the eye oscillations become more marked, and shock may develop.

### *Intense Illumination*

A level of illumination in excess of the amount needed for good vision may produce a feeling of discomfort and eye fatigue. An intensely brilliant light source such as the sun, carbon arc, or welder's arc may effect temporary or permanent blind areas in the retina. This occurs when the retina is subjected to intense light without proper protection and is known as *eclipse blindness*. Possibly this is due to the retinal heating effect of intense visible light and of infrared rays. *Snow blindness*, which exhibits some of the symptoms of visual blindness, is principally characterized by a burning and scratching of the external surfaces of the eye resulting from exposure to the ultraviolet portions of the spectrum, which are absorbed in these tissues. In this regard, snow blindness is the equivalent of a sunburn of the eyeball.

### *Glare*

Glare depends upon the general brightness of the field in which the glare source is seen. This is illustrated by the glare produced by a candle flame in a dark room, although the photometric brightness of the flame is not very great. The glare results because the eye is dark adapted to the decreased illumination of the whole field of view. There seems to be considerable individual variation with regard to susceptibility to glare. Prolonged exposure to an environment in which high contrasts of brightness produce glare may condition the observer so that he does not consider the glare to be as objectionable as before. Glare may produce a feeling of visual discomfort. If the glare is substantial or frequently induced it may effect tiredness, irritability, possibly headache, and a decrease in efficiency.

Glare may be produced directly by the light source itself, or indirectly by reflection of the light source from a shiny surface in the field of view such as shiny ceilings or walls, glass desk tops, polished metal surfaces, or glossy paper.

### *Color*

The color of a light source may be an important cause of defective illumination; however, in most working conditions color control is not so important as the proper selection of the light intensity or the elimination of glare. The spectral quality of light, however, is of major importance in some jobs including those involving color discrimination or color matching.

The painting of machinery and surroundings with carefully chosen colors has been introduced into industry to improve seeing and provide cheerful, pleasant, and interesting work environments.

### *Shadows*

Improper diffusion and distribution of light may cause shadows. This defect of illumination is an important cause of industrial accidents.

Artificial lighting in many modern factories and offices is accomplished by means of the fluorescent lamp. Special attention is necessary in planning a fluorescent lighting installation, especially on a moving production line or if revolving objects are to be in the field of view. This is important because the light from a fluorescent lamp varies rhythmically despite its apparent steadiness and may give rise to a stroboscopic effect, or an illusion of movement. Usually this effect can be eliminated by the proper grouping and installation of the fluorescent lamps.

### *Potential Occupational Exposures*

Virtually all occupations offer potential exposure to the hazards of defective illumination. Some occupations, however, are especially subject to this hazard. Such occupations usually require close fine work and attention to detail for many hours a day; for example, engravers, draftsmen, jewelers, watchmakers, miners, mail sorters, and clerks.

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## (6) Noise and Vibration

Advanced mechanization has created excessive noise and vibration conditions in many different occupational environments. While much is already known about the adverse effects of noise and vibration on man, more information will be required before specifications for tolerable noise or vibration conditions can be made on a valid basis. Presently, extensive efforts are being made to correlate various aspects of noise exposure with hearing loss in an attempt to verify proposed noise tolerance limits for protecting man's hearing. The potency of different vibration conditions for causing pain and discomfort to the individual has also been under study with regard to establishing safety limits for vibratory stimulation.

### Basic Aspects of Sound and Noise

Airborne sound refers to the rapid pressure variations, that is, alternate increases and decreases in normal atmospheric pressure, caused by a vibrating object and also the resultant sensation when such pressure changes strike the ear. The rate of vibration of the object corresponds to the frequency of sound, which is expressed in cycles per second (cps).

The frequency range of audible sounds for healthy young ears is usually considered to extend from 20 to 20,000 cps although there is evidence to indicate that man's hearing extends beyond these limits. The simplest type of sound, called a pure tone, is described as having a single frequency. These sounds are produced by tuning forks or electric means. In contrast, music, speech, and noise, each containing a collection of different frequency sounds, are called complex sounds. The frequencies comprising speech are found principally between 250 and 3,000 cps. This is considered to be the most important range of frequencies since hearing loss for speech sounds would handicap the individual in most daily activities.

Sound pressure level (SPL) measurements are based upon the average (root mean square) amplitude of the pressure changes constituting the sound

stimulus and are directly related to the intensity or energy characteristics of the sound. Such measurements are specified on a decibel scale and defined by the formula:

$$\text{SPL in decibels (db)} = 20 \log_{10} P/P_0$$

where  $P$  is a measure of the pressure change corresponding to the sound under study, and  $P_0$  is a reference pressure. The reference value for SPL determinations is usually 0.0002 dyne/cm<sup>2</sup>. This pressure corresponds to the weakest sound that the ear can hear under the most ideal listening conditions. SPL measurements must always state the reference value being used. Unless otherwise stipulated, all sound intensity values in this presentation refer to SPL values in db re 0.0002 dyne/cm<sup>2</sup>. The accompanying table shows SPL values for some everyday sounds.

Noise is commonly defined as unwanted sound and can be classified into three basic types, namely, wide-band noise, narrow-band noise, and impulse noise. The spectrum of a wide-band noise shows that the acoustical energy is distributed over a large range of frequencies. In determining such spectra, the noise is usually divided into eight frequency bands, each one octave wide, and SPL measurements are made in each band. Examples of wide-band noise can be found in the weaving room of a textile mill and in jet aircraft operations.

*SPL in decibels re 0.0002 dyne/cm<sup>2</sup> for everyday sounds*

Threshold for pain	—140—
Pneumatic chipper at 5 feet	—130—
Subway train at 20 feet	Boiler shop (maximum level)
Train whistle at 500 feet	—120—
Heavy traffic at 25–50 feet	—110—
Conversational speech at 3 feet	—100—
Light traffic at 100 feet	—90—
Most sensitive hearing threshold	—80—
	Inside motor bus
	Office with tabulating machines
	—70—
	Average traffic
	—60—
	Private business office
	Average residence
	—50—
	—40—
	—30—
	—20—
	—10—
	— 0—

Narrow-band noises have most of their energy confined to a narrow range of frequencies and normally produce a definite pitch sensation. Accurate spectral determinations of narrow-band noises require SPL measurements in frequency bands which are smaller than an octave in width. The noise caused by a circular saw, planer, or other power cutting tools are of the narrow-band type.

The impulse type of noise consists of transient pulses, occurring in repetitive or nonrepetitive fashion. Repetitive impulse noise is associated with the operation of a rivet gun or a pneumatic hammer. The impact of a drop hammer and the firing of a gun are examples of nonrepetitive impulse noise. The instrumentation, together with the procedures used to describe impulse noise, differs from that used for narrow or wide-band types of noise. Repetitive impulse noise which occurs at a rate exceeding 200 pulses per minute, however, can be analyzed in a manner similar to that used for wide- or narrow-band noise.

### *Effects of Noise on Hearing*

Exposure to intense noise causes hearing losses which may be temporary, permanent, or a combination of the two. These impairments are reflected by elevated thresholds of audibility for different frequency sounds, the increase in decibels required to hear such sounds being used as a measure of the loss. Temporary hearing losses, also called auditory fatigue, represent threshold losses which are recoverable after a period of time away from the noise. Such losses may occur after only a few minutes of exposure to intense noise. With prolonged exposures (months or years) to the same noise, there may be only partial recovery of the threshold losses, the residual loss being indicative of a permanent hearing impairment.

*Temporary hearing impairment*—Extensive studies have been made of temporary hearing losses following various conditions of noise exposure. Such investigations have yielded the following observations:

(1) Typical industrial noise exposures produce the largest temporary hearing losses at 4,000 and 6,000 cps. The greatest portion of the loss occurs within the first two hours of exposure. Recovery from such losses is greatest within one or two hours after exposure.

(2) The amount of temporary hearing loss from a given amount of noise varies considerably from individual to individual. Indeed, the losses at a given frequency from noise intensities of 100 db or more may range from 0 to more than 30 db for the exposed group. On the other hand, the amount of threshold loss for the group varies according to a normal statistical distribution; that is, few persons show very large or very small shifts, and most of the losses cluster around an average midway between the largest and smallest threshold shift.

(3) Low frequency octave bands of noise, below 300 cps, require considerably more intensity than middle or high frequency octave bands of

noise to produce significant threshold losses. The lowest intensity level capable of producing a temporary threshold loss is 80 db.

(4) Considerably fewer temporary hearing losses result from intermittent than from continuous noise exposures, even though the total amount of noise exposure is the same in both instances.

(5) The amount of temporary loss and its frequency location vary with the amount and frequency location of a permanent loss. Generally, the amount of temporary loss that occurs at a given frequency becomes less as the amount of permanent loss increases for that frequency.

It is presently believed that there is a direct relationship between temporary hearing loss and permanent hearing loss. A noise that does not cause temporary hearing loss following a short term exposure, for example, is assumed to be incapable of producing a permanent hearing impairment. Moreover, the pattern of temporary hearing losses shown for various frequency sounds following exposure to a given noise is assumed to resemble the pattern that will occur if and when a permanent loss is produced by a long-term exposure to the same noise. Recent findings concerning the nature of permanent noise-induced hearing loss provide some support for these assumptions and are contained in the discussion below.

*Permanent hearing loss*—Exposure to intense noise is only one cause of permanent hearing damage. Other causes may be disease, mechanical injury, and use of drugs. The time and nature of onset of the loss, the pattern of hearing loss for different frequencies, the findings of an otologic examination and medical history are means of determining whether a case of permanent hearing damage is due to these latter factors. Once these causes have been excluded from the etiology of hearing damage, the losses attributable to the aging process (presbycusis) must be considered. Curves showing the normal deterioration in hearing with increasing age have been reported. Such curves have been used to separate the amount of hearing loss due to noise exposure from that due to the aging process.

Evidence for permanent hearing losses from occupational noise has come from two basic types of study. In one, the hearing of personnel who work under characteristically noisy conditions, for example, factory workers, has been compared with that of a similar age group of persons who work under typically quiet conditions, such as office workers. In the other, pre-employment tests of hearing sensitivity have been compared with follow-up hearing tests for worker groups exposed to various amounts of noise. Suitable corrections are made in the latter type of study to account for hearing loss due to age only. The findings from both types of study show that permanent threshold losses caused by noise initially appear in the region 3,000 to 6,000 cps and are most prominent at 4,000 cps. With continued exposure, the losses become greater and spread to frequencies above and below the 3,000 to 6,000 cps range until the hearing at most frequencies is affected.

Since early noise-induced losses almost always occur at frequencies above the speech range, substantial impairments in hearing can occur without the individual's being aware of such damage. Actually, impairments in the perception of speech do not become noticeable until losses for the speech frequencies are 20 db or more.

The losses in hearing due to exposure to a given occupational noise tend to reach a maximum at certain frequencies (for example, 4,000 cps) after about 10 years of exposure; further losses in hearing at these frequencies appear to be accounted for by the aging process. The hearing loss for these frequencies which results from a 10-year exposure to noise for a worker appears to correlate closely with the temporary hearing loss at the same frequencies following a new worker's first day of exposure to such noise. The latter finding has particular significance for the assumed relationship between temporary and permanent hearing loss which was cited earlier. Indeed, it suggests the possibility of using temporary threshold losses as a susceptibility index for predicting permanent noise-induced hearing losses.

### *Damage Risk Criteria for Noise Exposure*

Protecting the worker against noise-induced losses in hearing requires specification of the boundary conditions or limits for safe noise exposures. Initially, proposals for noise tolerance limits were expressed solely in terms of an over-all level of noise intensity. It became quickly apparent, however, that the spectrum of the noise, the time distribution of the individual exposure periods, the total duration of exposure, and the susceptibility of the person exposed also had to be included in a statement of such limits. At present considerable disagreement is reflected in estimates of safe noise exposure limits.

### *Speech Interference and Nonauditory Effects of Noise*

*Interference with communication*—Noise which is not intense enough to cause hearing damage may still disrupt speech communication and the hearing of other desired sounds. Such disruptions will affect performance on those jobs which depend upon reliable speech communication. Even more important, however, is the fact that the inability to hear commands or danger signals due to excessive noise increases the probability of severe accidents.

The arithmetic average of the readings in decibels for the three octave bands (600–1,200 cps, 1,200–2,400 cps, and 2,400–4,800 cps) contained in wide-band noise can provide a simple indication of the ability of that noise to affect the intelligibility of person-to-person speech communication. This average is referred to as the *speech-interference-level* (SIL). In noises whose spectra yield an SIL of 75 db, personnel would have to speak in a very loud voice and use a selected and possibly prearranged vocabulary to be understood over a distance of 1 foot. Telephone use under these condi-

tions would probably be impossible. Noise having an SIL of 65 db would permit barely reliable communication with a raised voice over 2 feet. This range of communication would be extended to 4 feet by using a very loud voice, and to 8 feet by shouting. Telephone conversations under these SIL conditions would be difficult. In noise fields having an SIL of 55 db, a normal voice can communicate effectively over a distance of 3 feet, a raised voice over 6 feet, and a very loud voice over 12 feet. An SIL of 55 db would be permissible in work situations such as business or secretarial offices. When noise does not exceed an SIL of 45 db, a relaxed normal voice may be used for a distance of 10 feet. Such conditions would be ideal for private offices, or conference rooms.

Procedures for predicting speech communication under narrow-band noise conditions, under conditions where several conversations are going on simultaneously, and under peculiar reverberant room conditions have not as yet been completely worked out.

*Impairments to performance*—The effects of excessive noise on efficiency and work output seem to be somewhat slighter than is often thought. Performance on tasks involving simple repetitive operations, for example, does not appear to be affected by intense noise.

While efficiency in performing more complex operations may be adversely affected by noise, such effects in many instances tend to become dissipated as exposure time increases. One type of task, however, that shows pronounced and sustained performance decrements due to excessive noise is one requiring the worker to maintain a continuous watch over a number of dials to detect and report the presence of rarely occurring signals. This finding has implications for watch-keeping jobs where a worker may have to continuously scan or monitor a number of indicators to insure that no faults are developing in a machine or process. The finding also has practical importance for jobs requiring the inspection of items passing on a conveyor belt which cannot be viewed for an unlimited period of time.

The apparently limited effects of noise on performance make it difficult to determine which types of noise conditions will produce the greatest effects on work efficiency. The available evidence suggests that noises having over-all levels which exceed 90 db and containing predominantly high frequency components will be most effective in impairing performance.

*Annoyance*—Perhaps the most general reaction to noise is that of annoyance. Admittedly, there are wide individual differences as to what constitutes an annoying sound because of the many nonacoustical considerations that enter into such judgments. However, there are some basic characteristics of sound more annoying than others. These include:

(1) *Loudness*—the more intense and consequently louder noises are considered more annoying.

(2) *Pitch*—a high pitch noise, that is, one containing predominantly frequencies above 1,500 cps, is more annoying than a low pitch noise of equal loudness.

(3) *Intermittency and irregularity*—a sound that occurs randomly or varies in intensity or frequency is believed more annoying than one which is continuous and unchanging.

(4) *Localization*—a sound which repeatedly tends to change in localization is less preferred than one which remains stationary.

(5) *Inappropriateness to one's activity*—an example is the difference in attitude toward music when awake and when trying to sleep.

A measure of noise which is intended to describe its annoyance value has recently been developed and found to be successful in predicting the acceptability of fly-over noises produced by various types of aircraft. The measure is referred to as *perceived noise level in db* and is derived from calculations based upon the octave band intensity levels of the noise in question, together with data showing equal annoyance ratings for different octave bands of noise.

*Physiologic effects*—Physiologic reactions to a noise of sudden onset represent a typical startle pattern. There is a rise in blood pressure, an increase in sweating, an increase in heart rate, changes in breathing, and sharp contractions of the muscles over the whole body. These changes are often regarded as an emergency reaction of the body, increasing the effectiveness of any muscular exertion which may be required. However desirable in emergencies, these changes are not desirable for long periods since they would interfere with other necessary activities. Fortunately, these physiologic reactions subside with repeated presentations of the noise.

It has often been stated that for performance on a task to remain unimpaired by noise, man must exert greater effort than would be necessary under quiet conditions. Measures of energy expenditure—for example, oxygen consumption and pulse rate—show changes in the early stages of work under noisy conditions which are indicative of increased effort. With continued exposure, however, these responses return to their normal level.

### *Effects of Ultrasonic Stimulation*

Sounds whose frequencies are above the upper frequency limit of audibility are called ultrasonic. Ordinarily, ultrasonic sounds are defined as being in excess of 20,000 cps.

Interest in the possible harmful effects of ultrasonic sounds on man became highlighted when jet propulsive devices came into use. The noise spectra of these devices contained a broad range of ultrasonic frequencies which were initially believed to be the basis for the headaches, nausea, undue fatigue, dizziness, and other complaints reported by personnel who worked in the jet sound field. Subsequent research, however, indicated no support for this belief. It was suggested that the ill effects were more probably due to the

tremendous intensities of sound, over 140 db, created in the audible range of frequencies by the jet engines.

One of the best known effects of airborne ultrasonic radiation is the production of heat on the body's surface. On surfaces having a high coefficient of heat absorption such as furry animals, ultrasonic stimulation above 150 db in intensity can cause death through overheating. Much higher intensities of ultrasonic stimulation are needed to create a similar effect on man because of the comparatively low coefficient of heat absorption of the human skin and the ability of the body to throw off heat through its thermal regulatory mechanism. These factors, together with the losses in ultrasonic intensity due to attenuation in air, make it highly unlikely that an individual will be exposed to lethal doses of airborne ultrasonic radiation. High intensity ultrasonic stimulation focussed at specific areas of the human body, however, will cause localized tissue and cellular damage which is attributed to the heating effect. This type of stimulation has been useful in experiments aimed at identifying the functions of various tissues and cells through selective destruction techniques. It also has implications for the removal of tumors that might not be otherwise reached with usual surgical procedures.

When transmitted through a liquid, intense ultrasounds form cavities which, upon collapsing, produce shock waves strong enough to tear holes in metals and other solids. This property, called *cavitation*, has led to the use of ultrasonics in the cleaning of metals. It has also provided the means for mixing liquids which would not otherwise mix, and breaking up complex chemical compounds. Subjecting blood to the cavitation effect caused by ultrasonic stimulation can rupture the membranes of the red blood cells. The conditions required for cellular disintegration, however, are not found with the typical applications of ultrasound so that this hazard is considered as unlikely.

With the exception of experimental applications, present uses for ultrasound are believed to pose only a slight risk to the exposed individual. Future applications, however, may require higher intensity sources which can pose a more serious hazard to the operator, particularly if he has contact with the source or if the medium of ultrasonic transmission to the operator is other than air—for example, liquid or solid.

### *Characteristics of Vibratory Motion*

Vibration refers to any back-and-forth motion of matter. For present purposes, however, vibrations will refer only to low frequency back-and-forth motions of objects which are in contact with human beings. The vibratory range of particular interest to man is 1 to 400 cps, although it must be mentioned that the skin can detect vibrations in excess of 1,500 cps in frequency.

Besides frequency, other features of vibratory motion are displacement, velocity, and acceleration, each of which can serve to describe the magnitude

of vibratory motion. *Displacement* refers to the distance between the normal resting position of an object and its position at a given time in its vibratory cycle. The maximum displacement of the object from its normal resting position is called the amplitude of vibration. *Velocity* refers to the time rate of change of displacement. When a vibrating object reaches its peak upward or peak downward displacement, its velocity is zero. As the object passes in either an upward or downward direction through its normal resting position its velocity becomes maximal. The rate at which the velocity of the vibratory motion changes in direction (upward or downward) and magnitude defines the *acceleration* of the motion. Acceleration has been the measure most frequently used to describe the magnitude of vibratory motion since it is proportional to the forces contained in the motion and because it yields a smaller and consequently less cumbersome range of numerical values.

### *Effects of Whole Body Vibration on Man*

The effect of vibration upon the body is motion and relative displacement. If the vibration frequency is below 3 cps, the body moves as a unit and the adverse effects experienced are of the type associated with motion sickness. As the frequency of vibration is increased, various parts of the body tend to respond differentially to the fluctuating forces. Specific frequencies within the range 4 to 12 cps, for example, will cause the hips, shoulders, and abdominal parts to resonate, resulting in an amplification of their response to the imposed vibration. The direction of the vibration (whether applied vertically or longitudinally) and the position of the person (sitting or standing) will have some influence upon the amount as well as the specific frequencies of resonance for these body components. Between 20 and 30 cps the skull will begin to resonate, which leads to a deterioration in visual acuity. A similar disturbance will occur between 60 and 90 cps, when the eyeballs show a tendency to resonate with the vibrating forces.

Animal studies have shown that high amplitudes of whole body vibration (acceleration of 10 to 20g; g equals 32 ft. per sec. per sec.) for short durations may cause mechanical damage to the heart, lungs, brain, intestines and other parts of the abdominal region. These types of vibration-induced injury appear possible for man. It is too early, however, to speculate from the animal data as to what frequency, amplitude, and duration conditions will cause such effects in humans. It is also of importance to note that a person may suffer chronic injuries from vibration exposures of long duration which cause no apparent acute effects. These effects are found after exposure to repeated blows or to random jolts such as those experienced in riding vehicles. Buffeting in aircraft or high speed small craft on the water, or shaking in tractors or in other heavy vehicles on rough surfaces, gives rise to jolting motions. Acute injuries from these conditions are rare, but complaints of discomfort are common. Truck and tractor operators, for example, often

have sacroiliac strain. Minor kidney injuries are sometimes suspected, and traces of blood may, in rare cases, appear in the urine.

Subjective responses to whole body vibration include perception of motion, feeling of discomfort, apprehension, and pain. Such responses depend upon a number of factors including vibration frequency, acceleration and duration of exposure.

Physiologic reactions to vibration have not as yet been extensively studied. Changes in respiration, heart activity, and peripheral circulation have been found in response to vibration but appear to be of a transient nature. Certain postural reflexes, such as the knee jerk, appear to be inhibited by vibratory motion.

### *Localized Vibration Effects*

The best known of the injurious effects of vibration, and the one of immediate interest to industry, is associated with the use of hand-held power tools. Extensive use of pneumatic picks, hammers, and drills have been found to lead to a condition called *dead hand* or *white fingers*. This condition is characterized by numbness and blanching of the fingers with some loss of muscular control and reduction of sensitivity to heat, cold, and pain. Clinical findings show that the localized vibratory effect on the hands leads to pathologic changes in the vascular and nervous systems, and in the joints, tissues, and bones.

Heavy hand tools, such as pneumatic hammers and drills, produce vibrations which typically are below 60 cps but have peak accelerations which may be 100g or more. In contrast, light hand tools, such as those used in polishing and buffing, produce vibratory frequencies which range from 170 to 800 cps and have peak accelerations below 15g. The apparent differences in the vibration conditions for the heavy and light hand tools has led to some differences in the associated hand injuries. A comparison of the pathologic hand conditions caused by heavy low-speed hand tools and light high-speed hand tools is shown in the accompanying table.

Nature of hand impairment	Heavy, low-speed tools	Light, high-speed tools
Blanching of fingers.....	Characteristic.....	Absent.
Pain.....	Usually not a major complaint.	Major complaint.
Change in vascular tone.....	Not reported.....	Tone increased.
Swelling.....	Occasional.....	Frequent.
Degenerative changes in bone.....	Frequent.....	Absent.
Distribution of symptoms of neurovascular disturbance.	Usually in same hand as symptoms.	Usually in both hands.

*Note:* Duration of work prior to onset of impairment was several years in the case of heavy, low-speed tools; days to months in the case of light, high-speed tools.

Based on *Dart, 1946.*

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## • section X

### BIOLOGIC HAZARDS

MARCUS M. KEY, M.D.

Biologic agents as a cause of occupational disease have declined in importance since the advent of environmental sanitation and the anti-infection drugs. However, the danger is still present for several diseases, as is evidenced by the 15 to 20 cases of anthrax reported yearly in the United States, and the frequently observed elevated Brucella agglutination titers and Q fever complement-fixing antibody titers among certain occupational groups. So long as these diseases persist in the animal reservoir, there will be human cases.

Biologic agents may be conveniently classified as viral and rickettsial, bacterial, fungal, and parasitic. Potential occupational exposures are listed for the diseases in each of the four groups. For clinical manifestations and diagnostic tests the reader may consult items in the appended list of references.

#### Viruses and Rickettsia

Two important diseases in this group are ornithosis and Q fever. Though ornithosis was originally associated with the parrot family, it also affects ducks, chickens, and turkeys and has caused several outbreaks in poultry processing plants. The virus is present in nasal discharges and droppings of infected birds, and in aerosols from poultry processing. The virus enters man by the upper respiratory tract. Q fever is probably acquired by inhalation of the dried rickettsia in dairy or wool dust. Both of these infections have been acquired in the laboratory.

#### *Potential Occupational Exposures*

The following viral or rickettsial infections are potentially associated with the given occupations.

##### CATSCRATCH DISEASE

cat handlers  
dog handlers  
veterinarians

##### ECTHYMA CONTAGIOSUM (ORF)

ranchers  
sheep handlers  
shepherds

## MILKER'S NODULES

dairy workers  
farmers  
veterinarians

## NEWCASTLE DISEASE

chicken handlers  
laboratory workers  
poultry house workers  
poultry processors  
turkey handlers

## ORNITHOSIS (PSITTACOSIS)

canary handlers  
chicken handlers  
dock workers  
duck handlers  
laboratory workers  
lovebird handlers  
parakeet handlers  
parrot handlers  
pigeon handlers  
poultry processors  
turkey handlers

## Q FEVER

animal breeders  
cattle handlers  
dairy workers  
farmers  
goatherds  
laboratory workers  
shepherds  
slaughterhouse workers  
wool handlers

## RABIES

dog pound workers  
mail carriers  
meter readers  
veterinarians

## ROCKY MOUNTAIN SPOTTED FEVER

farmers  
foresters  
hunters  
laboratory workers  
ranchers  
shepherds  
trappers

## Bacteria

Secondarily infected occupational trauma or dermatitis may be found in almost any occupation. The causative organisms are usually staphylococci or streptococci. Insoluble cutting oils commonly cause folliculitis and furuncles, but the infection occurs from skin bacteria being trapped within the follicle rather than from pathogenic bacteria in the insoluble cutting oil.

Industrial anthrax is an infection acquired from infected wool, hair, or hides imported from countries where anthrax is present. It is usually cutaneous, but may be pulmonary when the spores are inhaled. Agricultural anthrax is acquired from infected livestock in certain enzootic areas of the United States.

*Potential Occupational Exposures*

The following bacterial infections are potentially associated with the given occupations.

## ANTHRAX (WOOLSORTER'S DISEASE)

animal handlers  
bacteriologists

blanket makers

bonemeal workers  
broom makers

## ANTHRAX (WOOLSORTER'S DISEASE)—con.

brush makers  
butchers  
carpet cleaners  
carpet makers  
cattle handlers  
cobblers  
cordage factory workers  
curriers  
dairy workers  
dock workers  
farmers  
fat renderers  
felt makers  
fertilizer makers  
freight handlers  
fur carders  
fur clippers  
fur cutters  
fur handlers  
fur preparers  
fur pullers  
gelatin makers  
glue makers  
goat hair handlers  
goat hide handlers  
hair workers  
leather workers  
mattress makers  
meat inspectors  
minkery workers  
plasterers  
ragmen  
shavers, felt hat  
shavers, fur  
shaving brush makers  
shepherds  
slaughterhouse workers  
stablemen  
tanners  
tannery workers  
taxidermists  
upholsterers

veterinarians  
warehouse workers  
wooden heel workers  
wool carders  
wool scourers  
wool spinners  
wool workers

## BRUCELLOSIS (UNDULANT FEVER)

butchers  
carcass handlers  
cattle handlers  
cooks  
dairy workers  
farmers  
gardeners  
goatherds  
hide handlers  
manure handlers  
meat inspectors  
milk inspectors  
packing-house workers  
sausage stuffers  
sewer workers  
shearers  
shepherds  
slaughterhouse workers  
stablemen  
stockmen  
swine handlers  
veterinarians  
zoologic technicians

## ERYSYPELOID

butchers  
button makers, bone  
cooks  
farmers  
fishermen  
fish handlers  
game handlers  
kitchen workers  
meat inspectors  
swine handlers  
veterinarians

**FOLLICULITIS, FURUNCULOSIS**

animal handlers  
 battery makers, storage  
 chocolate workers  
 glue workers  
 hospital attendants  
 ice cream workers  
 machinists  
 mechanics  
 oilers  
 pitch workers  
 sugar workers  
 tallow refiners  
 tar workers  
 veterinarians

**LEPTOSPIROSIS (WEIL'S DISEASE)**

animal handlers  
 canal workers  
 cane field workers  
 cattle handlers  
 dairy workers  
 ditch diggers  
 dock workers  
 dog pound workers  
 farmers  
 fishermen  
 fish market workers  
 gardeners  
 miners  
 pig farm workers  
 poultry dressers  
 rice field workers  
 sewer workers  
 slaughterhouse workers  
 street cleaners  
 swine handlers  
 trench diggers

**tunnel diggers**

veterinarians

**LISTERIOSIS**

animal handlers  
 cattle handlers  
 dairy workers  
 sheep handlers

**PLAQUE**

geologists  
 hunters  
 linemen  
 shepherds

**TETANUS**

butchers  
 cattle handlers  
 farmers  
 horse handlers  
 packing-house workers  
 slaughterhouse workers

**TULAREMIA**

bacteriologists  
 butchers  
 cooks  
 farmers  
 forestry workers  
 hunters  
 rabbit handlers  
 shearers

**VERRUCA NECROGENICA**

anatomists  
 autopsy room attendants  
 butchers  
 dissecting room attendants  
 embalmers  
 nurses  
 physicians  
 slaughterhouse workers

## Fungi

A wide variety of fungi is responsible for mycotic infections. Many of these infections are superficial and localized, and of minor clinical importance; others are systemic and involve deeper tissues, with occasional fatal outcome.

Dermatophytosis or athlete's foot is not usually an occupational disease. It is generally accepted that the causative dermatophytes are not acquired from the floors of showers and locker rooms, but are carried on the skin of most persons without causing disease. The cause is a lowering of local resistance by occlusive footwear, increased perspiration, and poor foot hygiene in certain susceptible individuals.

Occupationally-caused fungous diseases of the lungs include histoplasmosis, coccidioidomycosis, and aspergillosis. The symptoms and chest findings are similar to those associated with tuberculosis, but sputum and immunologic studies help to differentiate these diseases. Blastomycosis is sometimes classified as an occupational disease of farmers. This disease, as well as histoplasmosis and coccidioidomycosis, is limited to certain endemic areas.

### *Potential Occupational Exposures*

The following fungal infections are potentially associated with the given occupations

ASPERGILLOSIS	laborers
bird handlers	
grain mill workers	
CHROMOBLASTOMYCOSIS	
farmers	
laborers	
COCCIDIODOMYCOSIS	
farmers	
fruit pickers	
shepherds	
HISTOPLASMOSIS	
farmers	
guano workers	
poultrymen	
MONILIASIS	
bakers	
bartenders	
cannery workers	
dishwashers	
kitchen workers	
MYCETOMA PEDIS	
farmers	
SPOROTRICHOSIS	
berry pickers	
farmers	
florists	
foresters	
gardeners	
nurserymen	
South African miners	
TINEA CIRCINATA (RINGWORM)	
animal handlers	
barbers	
cat handlers	
cattle handlers	
dog handlers	
farmers	
fur handlers	
hide bundlers	
horse handlers	
monkey handlers	
wool sorters	

## Parasites

Many vegetables, fruits, and food products have associated mites which may fortuitously attack man. These mites may also parasitize rats and several species of birds.

Creeping eruption is limited to the southeastern United States, where the warm, sandy soil favors the hatching out of infective larvae from dog and cat hookworm eggs.

### Potential Occupational Exposures

The following parasitic infections or infestations are potentially associated with the given occupations.

CHIGGER BITES	cotton seed handlers
construction workers	farmers
farmers	grain elevator workers
linemen	strawboard makers
pipeline workers	
surveyors	
CREEPING ERUPTION	GROCER'S ITCH
brick masons	cheese handlers
ditch diggers	copra handlers
laborers	date handlers
lifeguards	dock workers
plumbers	fig handlers
	flour handlers
FOWL MITE DERMATITIS	grocers
office workers	meal handlers
poultrymen	prune handlers
	vanilla handlers
GRAIN ITCH	
barley handlers	

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## • section XI

### PLANT AND WOOD HAZARDS

MARCUS M. KEY, M.D.

Many plants and plant products, as well as woods, are known to cause occupational diseases with local and systemic manifestations. Plants and plant products are common offending agents producing these manifestations, dermatitis being the most frequently observed effect.

#### Plants and Plant Products

The most common occupational disease from plants is allergic contact dermatitis from poison ivy and oak which usually has such characteristic linear and bullous lesions that it is easily recognized as of plant origin. Poison ivy thrives throughout the United States, except in the southwest, and causes contact dermatitis in a high percentage of outdoor workers. Poison oak is equally troublesome, but is restricted to the west coast. A variant of poison ivy, called oakleaf poison ivy, is found in the southeastern United States.

Poison ivy and oak belong to the Anacardiaceae, or cashew, family of plants which includes other sources of sensitizing or cross-reacting agents, such as poison sumac, mango, Japanese lac tree, Indian marking nut, and cashew nut tree. From cashew nut shells is extracted a phenolic liquid which forms a condensation resin when combined with formalin. It is known as cashew nut shell liquid-formaldehyde resin and is used in the manufacture of varnishes and brake shoe linings. The liquid is a potent sensitizer as well as a strong primary irritant.

In the southern United States, the bastard feverfew, a common weed, produces an allergic contact dermatitis similar to that caused by poison ivy.

The harvesting and processing of fruits and vegetables are attended by allergic contact dermatitis. This has been reported from handling asparagus, carrots, oranges, and lemons, among others. In addition, fruit and vegetable handlers may also develop contact dermatitis from insecticides and fungicides; severe chapping and moniliasis from excessive exposure to

moisture; photosensitization dermatitis from concurrent or subsequent exposure to sunlight; and parasitism from exposure to fruit, vegetable, and grain mites.

Frequent plant photosensitizers are fig, rue, lime, bergamot, and members of the Umbelliferae including parsnip, parsley, carrot, fennel, dill, and celery (pink rot). By photosensitization is meant the delayed development of erythema, edema, vesicles, and bullae following contact with the plant juices and exposure to sunlight. This is an accentuated localized sunburn, and eventuates in either hyperpigmentation or depigmentation, depending on the severity of the reaction. Reported case of phytophotosensitization have been instances of phototoxicity rather than photoallergy. Phytophototoxins are psoralen compounds, and their activity is believed to be associated with the furocoumarin ring.

Contact with certain flowers frequently produces dermatitis on an allergic basis. Examples are chrysanthemum, pyrethrum, primrose, and the bulbs of narcissus, tulips, and hyacinth. Ragweed pollen may cause several types of occupational allergy among farmers and highway workers. The water-soluble protein fraction of the pollen may cause hay fever and asthma. The lipid fraction may cause an eczematous dermatitis of exposed areas.

Reports of asthma, hay fever, and urticaria from castor bean processing are common. The dried pomace resulting from castor oil extraction contains a potent allergen which is responsible for symptoms reported among castor bean workers and others in the vicinity of extraction plants, among farmers using the pomace as fertilizer, and among dock workers engaged in unloading bags of castor bean pomace.

### *Potential Occupational Exposures*

Botanists	Fruit processors
Bulb handlers, plant	Gardeners
Camp workers	Highway workers
Canners	Hop pickers
Construction workers	Horticulturists
Dock workers	Pipeline workers
Farmers	Road builders
Field laborers	Surveyors
Flower cutters	Telephone linemen
Flower packers	Utility workers
Foresters	Vegetable harvesters
Fruit pickers	Vegetable processors

### Woods

Occupational diseases from woods may be classified as toxic, irritant, or allergenic in nature.

A toxic wood is one that contains a substance, usually an alkaloid, which may be inhaled, ingested, or absorbed through the skin with the resultant occurrence of systemic signs and symptoms. These may include headache, anorexia, nausea, vomiting, bradycardia, dyspnea, and somnolence. Examples of toxic woods are East Indian satinwood, South African boxwood, and ipe.

An irritant wood may cause injury to mucous membranes when contact occurs and symptoms of sneezing, coughing, rhinorrhea, or tearing may result. Some irritant woods may damage the intact skin resulting in contact dermatitis of the primary irritation type. *Mansonia*, *dahoma*, and *cocobolo* are examples of irritant woods.

An allergenic wood may precipitate many different allergic manifestations in a sensitized individual. Most common of these are asthma and contact dermatitis. Certain members of the birch, pine, dogwood, beech, mahogany, mulberry, and myrtle families present examples of such woods.

Patch testing with sawdust, shavings, and sap may aid in differentiating between primary skin irritation and skin sensitization.

### *Potential Occupational Exposures*

Cabinet makers	Musicians
Carpenters	Sawmill operators
Lumbermen	Violin makers

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## • section XII

### SOURCES OF CONSULTATION ON OCCUPATIONAL HEALTH

The problem of a possible relationship between the patient's illness and his occupational environment is not always easily solved. At times the investigator may feel the need of consultation after he has reviewed the pertinent literature and given consideration to all of the factors believed relevant. Sources of consultation reside in the official and nonofficial agencies. The official agencies include those established in the Federal Government and those existing in States, counties and cities. Among the nonofficial agencies are the insurance companies, particularly those issuing workmen's compensation insurance policies, a number of universities, and professional groups engaged in private consultation. This section presents a list of the State and local agencies with resources available to the investigator, the diversity and extent of such resources being dependent upon administrative and budgetary factors and the supply of trained personnel.

#### State and Local Agencies

**ALASKA** Environmental Health, Division of Public Health, Alaska Department of Health and Welfare, Alaska Office Building, Juneau 99801

**CALIFORNIA** (a) Bureau of Occupational Health, California Department of Public Health, 2151 Berkeley Way, Berkeley 94704. (b) Division of Industrial Safety, Department of Industrial Relations, 455 Golden Gate Avenue, San Francisco 94102. (c) State Compensation Insurance Fund, Department of Industrial Relations, 525 Golden Gate Avenue, San Francisco 94102

*Alameda County* Bureau of Environmental Health, Alameda County Health Department, 499 Fifth Street, Oakland 94607

*Albany City* Albany City Health Department, 1045 Solano Avenue, Albany 94706

*Berkeley City* Berkeley City Health Department, 2121 McKinley Street, Berkeley 94704

*Contra Costa County* Occupational Health Division, Contra Costa County Health Department, P.O. Box 871, Martinez

*Long Beach City* Long Beach City Health Department, 2655 Pine Avenue, Long Beach

*Los Angeles City* Division of Occupational and Radiological Health, Los Angeles City Health Department, 111 East First Street, Los Angeles 90012

*Los Angeles County* Division of Industrial Health and Hygiene, Los Angeles County Health Department, 241 North Figueroa Street, Los Angeles 90012

*Orange County* Division of Sanitation, Orange County Health Department, P.O. Box 355, Santa Ana 92702

*Pasadena City* Occupational and Radiological Health Services Section, Environmental Health Division, City of Pasadena, Department of Public Health, 100 North Garfield Avenue, Pasadena 91109

*San Bernardino County* Division of Environmental Sanitation, San Bernardino County Health Department, 316 Mountain View Avenue, San Bernardino

*San Diego County* Bureau of Industrial Hygiene and Air Pollution Control, County of San Diego Department of Public Health, Civic Center, San Diego 92101

*San Jose City* San Jose City Health Department, 151 West Mission, San Jose

*San Mateo County* Sanitation Section, San Mateo County Department of Public Health and Welfare, 225 37th Avenue, San Mateo

*Santa Clara County* Bureau of Occupational Health, County of Santa Clara Health Department, 2220 Moorpark Avenue, San Jose

*Santa Cruz County* Environmental Health Section, Santa Cruz County Health Department, P.O. Box 962, Santa Cruz

*Stanislaus County* Stanislaus County Health Department, 810 Scenic Drive, Modesto

*Vernon City* Vernon City Health Department, 4305 Santa Fe Avenue, Vernon

**COLORADO** Division of Occupational and Radiological Health, Colorado State Department of Public Health, 4210 East 11th Avenue, Denver 80220

*Denver City* Occupational Health Section, Environmental Health Service, Department of Health and Hospitals, 659 Cherokee Street, Denver 80204

**CONNECTICUT** Occupational Health Section, Division of Medical Services, Connecticut State Department of Health, State Office Building, Hartford 06115

**FLORIDA** Division of Radiological and Occupational Health, Bureau of Preventable Diseases, Florida State Board of Health, P.O. Box 210, Jacksonville 32201

*Hillsborough County* Division of Occupational and Radiological Health, Hillsborough County Health Department, P.O. Box 1731, Tampa 33601

GEORGIA Occupational Health Service, Georgia Department of Public Health, Atlanta 30303

*Fulton County* Industrial Sanitation Section, Fulton County Health Department, 99 Butler Street, S.W., Atlanta

HAWAII Occupational and Radiological Health Section, Health Engineering Branch, Division of Environmental Health, Department of Health, P.O. Box 3378, Honolulu

IDAHO Engineering and Sanitation Division, Idaho Department of Health, Statehouse, Boise 83701

ILLINOIS Industrial Hygiene Unit, Safety Inspection and Education Division, Illinois Department of Labor, 160 North LaSalle Street, Chicago

INDIANA Division of Industrial Hygiene, Indiana State Board of Health, 1330 West Michigan Street, Indianapolis

KANSAS Industrial, Radiation and Air Hygiene Program, Kansas State Department of Health, State Office Building, Topeka

*Wichita-Sedgwick County* Division of Environmental Health, Wichita-Sedgwick County Department of Public Health, 1900 East Ninth, Wichita 67214

KENTUCKY Occupational Health Program, Division of Environmental Health, Kentucky Department of Health, 275 East Main Street, Frankfort 40601

LOUISIANA Occupational Health and Safety Section, Division of Preventive Medicine, Louisiana State Board of Health, P.O. Box 60630, New Orleans

MAINE Occupational and Radiological Health Section, Division of Sanitary Engineering, State Department of Health and Welfare, Augusta 04330

MARYLAND Division of Occupational Health, State Department of Health, State Office Building, 301 West Preston Street, Baltimore

*Baltimore* Bureau of Industrial Hygiene, Baltimore City Health Department, 602 American Building, Baltimore 21202

MASSACHUSETTS Division of Occupational Hygiene, Massachusetts Department of Labor and Industries, 286 Congress Street, Boston 02210

MICHIGAN Division of Occupational Health, Michigan Department of Health, 3500 North Logan Street, Lansing 48914

*Detroit* Bureau of Industrial Hygiene, Detroit Department of Health, 8801 John C. Lodge Expressway, Detroit 48202

MINNESOTA Section of Occupational Health, Division of Environmental Health, Minnesota Department of Health, University Campus, Minneapolis 55440

*Minneapolis* Occupational Health Service, Division of Public Health, 250 Fourth Street South, Minneapolis 55415

MISSISSIPPI Division of Occupational Health, Mississippi State Board of Health, P.O. Box 1700, Jackson

MISSOURI Radiological and Occupational Health, Division of Health of Missouri, Jefferson City

*St. Louis City* Industrial Hygiene Section, St. Louis Division of Health, 62 Municipal Courts Building, St. Louis

MONTANA Division of Disease Control, State Board of Health, Helena

NEW HAMPSHIRE Occupational Health Service, Division of Public Health, Department of Health and Welfare, 61 South Spring Street, Concord

NEW JERSEY Occupational Health Program, Division of Environmental Health, New Jersey State Department of Health, 17 West State Street, Trenton 08625

NEW MEXICO Occupational Health Section, Division of Environmental Sanitation Services, New Mexico Department of Public Health, Santa Fe

*Albuquerque* Albuquerque City Health Department, P.O. Box 1293, Albuquerque 87103

NEW YORK Division of Industrial Hygiene, New York State Department of Labor, 80 Centre Street, New York 10013

NORTH CAROLINA Occupational Health Section, North Carolina State Board of Health, Raleigh

*Charlotte* Division of Environmental Health, City of Charlotte Health Department, 1200 Blythe Boulevard, Charlotte

OHIO (a) Division of Industrial Hygiene, Ohio Department of Health, 1147 Chesapeake Avenue, Columbus 43212. (b) Division of Safety and Hygiene, Industrial Commission of Ohio, 700 W. 3rd Avenue, Columbus

*Cincinnati* Occupational Health Services, Cincinnati Health Department, City Hall, Cincinnati

*Cleveland* Bureau of Industrial Hygiene, Division of Air Pollution Control, Cleveland Department of Urban Renewal and Housing, 14101 Lakeshore Boulevard, Cleveland 44110

OKLAHOMA Occupational and Radiological Health Section, Environmental Health Service, Oklahoma State Department of Health, 3400 North Eastern, Oklahoma City 73105

OREGON Occupational and Radiological Health Section, Division of Preventive Medical Services, Oregon State Board of Health, 1400 S.W. Fifth Avenue, Portland 97207

PENNSYLVANIA Division of Occupational Health, Pennsylvania Department of Health, Health and Welfare Building, Harrisburg 17120

*Allegheny County* Division of Occupational Health, Allegheny County Health Department, 620 City-County Building, Pittsburgh 15219

*Philadelphia* Occupational and Radiological Health Section, Division of Environmental Health, Philadelphia Department of Public Health, 500 South Broad Street, Philadelphia 19146

PUERTO RICO Section of Occupational Health, Division of Sanitation, Puerto Rico Department of Health, San Juan

RHODE ISLAND Division of Industrial Hygiene, Rhode Island Department of Health, 365 State Office Building, Providence

SOUTH DAKOTA Occupational and Radiological Health Section, Division of Sanitary Engineering; State Department of Health, Pierre

TENNESSEE Industrial Hygiene Service, Division of Preventable Diseases, Tennessee Department of Public Health, Cordell Hull State Office Building, Nashville 37219

TEXAS Division of Occupational Health and Radiation Control, Texas State Department of Health, 1100 West 49th Street, Austin

*Dallas* City of Dallas, Public Health Department, Dallas

*Harris County* Harris County Health Department, P.O. Box 4116, Houston 77014

*Houston* Industrial Hygiene Program, Houston City Health Department, 612 Bagby, Houston 77002

UTAH Section of Industrial Hygiene, Division of Sanitation, Utah State Department of Health, 45 S. Fort Douglas Boulevard, Salt Lake City 84113

VERMONT Industrial Hygiene Division, Vermont Department of Health, P.O. Box 333, Barre 05641

VIRGINIA Bureau of Industrial Hygiene, Virginia State Department of Health, State Office Building, Richmond 23219

WASHINGTON Division of Safety, Department of Labor and Industries, General Administration Building, Olympia 98502

WEST VIRGINIA Bureau of Industrial Hygiene, West Virginia Department of Health, State Office Building, Charleston

WISCONSIN Occupational Health Division, Wisconsin State Board of Health, State Office Building, 1 West Wilson Street, Madison

*Milwaukee* Industrial Hygiene Section, Environmental Technical Services Division, City of Milwaukee Health Department, 841 North Broadway, Milwaukee 53202

WYOMING Division of Industrial Hygiene, Wyoming Department of Public Health, State Office Building, Cheyenne



## • section XIII

### OCCUPATIONAL HEALTH REFERENCE AIDS

The occupational health reference aids listed in this section supplement the references appended to other sections. The material includes indexes, abstract journals and abstracting services, occupational health journals, bibliographies, and texts.

Separately presented is a selection of texts published in 1943 or earlier. An examination of these texts will reveal that some so-called modern practices date back many years. In short, the inclusion of the older works will have served its purpose if the reader sampling them comes to recognize that they do not deserve the oblivion to which many of them have been carelessly consigned. It should be pointed out, moreover, that the older literature is by no means confined to the recent past. The literature of previous centuries is relatively vast. The historically-minded reader may gain supplementary knowledge as well as stimulation by using as a guide the *Index-Catalogue of the Library of the Surgeon General's Office, U.S. Army*, particularly volume 10 published in 1889. The caption, "Occupations and trades (diseases and hygiene of)," requires seven quarto pages (67-73) to index the books and the articles carried by journals on the shelves of the library (now the National Library of Medicine, U.S. Public Health Service).

#### Indexes

**CHEMICAL TITLES**, Current Author and Keyword Indexes from Selected Chemical Journals, a Product of the Chemical Abstracts Service. American Chemical Society, Easton, Pa. Titles chosen from some 600 journals of pure and applied chemistry and chemical engineering are covered in semimonthly issues.

**INDEX-CATALOGUE** of the Library of the Surgeon General's Office, U.S. Army. Vol. 10. U.S. Government Printing Office, Washington, D.C., 1889.

**INDEX MEDICUS**, formerly *Current List of Medical Literature*. National Library of Medicine, U.S. Public Health Service, Washington, D.C. Indexes by subject and by author articles appearing in world medical literature. Published monthly. U.S. Government Printing Office, Washington, D.C. American Medical Association, Chicago, publishes annual cumulations under the title, *Cumulated Index Medicus*. Volume 1 covering 1960 was published in 1961 in three separately bound parts. Part 1 carries a list of the journals indexed as well as an author index. Parts 2 and 3 constitute a subject index.

### *Abstract Journals and Abstracting Services*

**APCA ABSTRACTS.** Air Pollution Control Association in cooperation with U.S. Public Health Service and Library of Congress. The Association, Mellon Institute, Pittsburgh, Pa. Abstracts prepared from some 500 journals appear monthly.

**BIOLOGICAL ABSTRACTS.** Biological Abstracts, Inc., University of Pennsylvania, Philadelphia. Abstracts covering the areas of biology, zoology and botany are published in semimonthly issues.

**BULLETIN OF HYGIENE.** Bureau of Hygiene & Tropical Diseases, Keppel Street, London. Carries a section on occupational hygiene. Presents abstracts of articles from the world literature. Published monthly in 2 editions, one printed on both sides of the page and the other on one side only.

**CHEMICAL ABSTRACTS,** Key to the World's Chemical Literature, a Product of the Chemical Abstracts Service. American Chemical Society, Easton, Pa. Published bi-weekly.

**CIS,** International Abstracting and Information Service on Occupational Safety and Health. International Labor Office, Geneva, Switzerland. Abstracts of articles on occupational safety and health appearing in the world literature are distributed monthly to subscribers on 3- by 5-inch cards.

**DIGEST OF NEUROLOGY AND PSYCHIATRY;** Abstracts and Reviews of Selected Literature in Psychiatry, Neurology and their Allied Fields. The Institute of Living, Hartford, Conn. Published monthly.

**EXCERPTA MEDICA,** International Medical Abstracting Service. Excerpta Medica Foundation, 2 East 103rd Street, New York, N.Y. Section 17 entitled Public Health, Social Medicine and Hygiene (including Industrial Medicine and Hygiene) is published monthly.

**INDUSTRIAL HYGIENE DIGEST.** Industrial Hygiene Foundation, Mellon Institute, Pittsburgh, Pa. Abstracts prepared from some 400 journals appear monthly.

**NUCLEAR SCIENCE ABSTRACTS.** U.S. Atomic Energy Commission, Oak Ridge, Tenn. Abstracts and indexes the literature of nuclear science and technology. Published twice monthly. U.S. Government Printing Office, Washington, D.C.

**PUBLIC HEALTH ENGINEERING ABSTRACTS.** Robert A. Taft Sanitary Engineering Center, U.S. Public Health Service, Cincinnati, Ohio. Includes abstracts on occupational health, radiologic health, and insect-borne diseases. Published monthly. U.S. Government Printing Office, Washington, D.C.

**U.S. GOVERNMENT RESEARCH REPORTS.** Office of Technical Services, U.S. Department of Commerce, Washington, D.C. Abstracts of research and development reports of the Army, Navy, Air Force, Atomic Energy Commission and other agencies of the U.S. Government. Published twice monthly. U.S. Government Printing Office, Washington, D.C.

### *Occupational Health Journals*

**AMERICAN INDUSTRIAL HYGIENE ASSOCIATION JOURNAL.** American Industrial Hygiene Association, 14125 Prevost, Detroit, Mich.

**ANNALS OF OCCUPATIONAL HYGIENE** (London). Pergamon Press, 122 East 55th Street, New York, N.Y.

**ARCHIVES BELGES DE MÉDECINE SOCIALE, HYGIÈNE, MÉDECINE DU TRAVAIL ET MÉDECINE LÉGALE.** Ministère de la Santé Publique et de la Famille, 43 Avenue des Arts, Brussels, Belgium

**ARCHIVES OF ENVIRONMENTAL HEALTH.** American Medical Association, 535 North Dearborn Street, Chicago, Ill.

*ARCHIVES DES MALADIES PROFESSIONNELLES, DE MÉDECINE DU TRAVAIL ET DE SÉCURITÉ SOCIALE.* Masson et Cie, Libraires de L'Académie de Médecine, 120 Boulevard Saint-Germain, Paris, France

*ARCHIV FÜR GEWERBEPATHOLOGIE UND GEWERBEHYGIENE.* Springer-Verlag, Heidelberger Platz 3, Berlin-Wilmersdorf, Germany

*BERUFSDERMATOSEN.* Editio Cantor, Verlag für Medizin und Naturwissenschaften, Aulendorf, Württemberg, Germany

*BRITISH JOURNAL OF INDUSTRIAL MEDICINE.* British Medical Association, Tavistock Square, London, England.

*INDUSTRIAL MEDICINE AND SURGERY.* Industrial Medicine Publishing Co., P.O. Box 44-306, Miami, Fla.

*JOURNAL OF OCCUPATIONAL MEDICINE.* Hoeber Medical Division of Harper & Row, 2 Park Avenue, New York, N.Y.

*LA MEDICINA DEL LAVORO.* Via S. Barnaba 8, Milan, Italy

*TRANSACTIONS OF THE ASSOCIATION OF INDUSTRIAL MEDICAL OFFICERS.* Association of Industrial Medical Officers, 47 Lincoln's Inn Fields, London, England

*ZEITSCHRIFT FÜR UNFALLMEDIZIN UND BERUFSKRANKHEITEN.* Buchdruckerei Berichthaus, Zwingliplatz 3, Zürich, Switzerland

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Index entries are primarily occupations and occupational disease agents. Included also are diseases, signs, and toxicologic terms. Numbers refer to pages.

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